IYKRWIILGLNKIVRMYSPT

QUERY	IYKRWIILGLNKIVRMYSPT
CONSENSUS_A	-------------------V
A.KE.Q23-CXC-CG	-------------------V
A.SE.SE6594	-------------------V
A.SE.SE7253	-------------------V
A.SE.SE7535	-------------------V
A.SE.SE8131	-------------------V
A.SE.SE8538	-------------------V
A.SE.SE8891	-------------------V
A.UG.92UG037	-----M-------------V
A.UG.U455	--R----------------V
CONSENSUS_B	--------------------
B.AU.AF128998	-------------------V
B.-.NL43E9	--------------------
B.AU.MBC18	-------------------I
B.AU.MBC200	--------------------
B.AU.MBC925	--------------------
B.AU.MBCC54	--------------------
B.AU.MBCC98	----------I---------
B.AU.MBCD36	--------------------
B.CN.RL42	--------------------
B.DE.D31	--------------------
B.DE.HAN	--------------------
B.ES.89SP061	--------------------
B.FR.HXB2	--------------------
B.GA.OYI	--------------------
B.GB.CAM1	--------------------
B.GB.MANC	--------------------
B.JP.JH31	-------------------S
B.NL.3202A21	--------------------
B.TW.LM49	--------------------
B.US.85WCIPR54	--------------------
B.US.AD8	--------------------
B.US.BC	-------------------S
B.US.DH123	-------M------------
B.US.JRCSF	-------------------V
B.US.JRFL	--------------------
B.US.MNCG	-------------------S
B.US.NC7	-------------------I
B.US.NY5CG	--------------------
B.US.P896	-------------------S
B.US.RF	-------------------I
B.US.SF2	--------------------
B.US.WC001	-------------------A
B.US.WEAU160	-------------------V
B.US.WR27	--------------------
B.US.YU2	--------------------
CONSENSUS_C	-------------------V
C.BR.92BR025	-------------------V
C.BW.96BW01B22	-------------------V
C.BW.96BW0402	-------------------V
C.BW.96BW0502	-------------------V
C.BW.96BW1104	-------------------V
C.BW.96BW1210	-------------------V
C.BW.96BW15B03	-------------------V
C.BW.96BW1626	-------------------V
C.BW.96BW17A09	------TM-----------V
C.ET.ETH2220	-------------------V
C.IN.93IN904	-------------------V
C.IN.93IN905	-------------------V
C.IN.93IN999	-------------------V
C.IN.94IN11246	-------------------V
C.IN.95IN21068	-------------------V
CONSENSUS_D	-------------------V
D.CD.84ZR085	-------------------V
D.CD.ELI	-------V-----------V
D.CD.NDK	-------------------V
D.CD.Z2Z6	-------------------V
D.UG.94UG1141	-------------------V
CONSENSUS_F	-------------------V
F.BR.BZ162	-------------------V
F.CD.VI174	-------------------V
F.RW.VI69	-------------------V
CONSENSUS_F1	-------------------V
F1.BE.VI850	-------------------V
F1.BR.93BR020.1	M------------------V
F1.FI.FIN9363	-------------------V
F1.FR.MP411	-------------------V
CONSENSUS_F2	-------------------V
F2.CM.MP255	-------------------V
F2.CM.MP257	-------------------V
CONSENSUS_G	--------x----------V
G.BE.DRCBL	-------------------V
G.FI.HH8793	-------------------V
G.NG.92NG083	-------------------V
G.SE.SE6165	-------------------V
CONSENSUS_H	-------------------V
H.BE.VI991	-------------------V
H.BE.VI997	-------------------V
H.CF.90CF056	-------------------V
CONSENSUS_J	-------------------V
J.SE.SE9173	-------------------V
J.SE.SE9280	-------------------V
CONSENSUS_K	-------------------V
K.BE.VI325	-------------------V
K.CD.EQTB11C	-------------------V
K.CM.MP535	-------------------V
N.CM.YBF30	--R---V----R-------V
CONSENSUS_O	--RK--V-----M-K----V
O.CM.ANT70C	--RK--V-----M-K----V
O.CM.MVP5180	--RK--V-----M-K----V
CRF01-AE.CF.90CF40	---K---------------V
CRF01-AE.TH.93TH25	-----------------Q-V
CRF01-AE.TH.CM240	-------------------V
CRF01-AE.TH.TH022	-------------------V
CRF01-AE.TH.TH047	-------------------V
CRF02_AG.FR.DJ263	-------------------V
CRF02_AG.FR.DJ264	-------------------V
CRF02_AG.NG.IBNG	------V-----------V
CRF03_AB.RU.KAL15	-------------------V
CRF04_cpx.CY.94CY0	---------------T---I
CRF04_cpx.GR.97PVC	-------------------V
CRF04_cpx.GR.97PVM	-------------------V
AC.ET.E3099G	-------------------V
AC.IN.21301	-------------------V
AC.RW.92RW009	-------------------V
AC.SE.SE9488	-------------------V
AC.ZM.ZAM174-21	-------------------V
AC.ZM.ZAM184	-------------------V
AC.ZM.ZAM716-17	-------------------V
ACD.SE.SE8603	-------------------V
AD.SE.SE6954	-------------------V
AD.SE.SE7108	-------------------V
ADHU.NO.NOGIL3	-------------------V
ADU.CD.MAL	-------------------V
AG.NG.G3	-------------------V
AG.SE.SE7812	------V-----------V
AGHU.GA.VI354	-------------------V
AGJ.AU.BFP90	-------------------V
AGJ.ML.95ML8	-------------------V
AGU.CD.Z321	-------------------V
BF.BR.93BR029.4	--------------------
DF.CD.VI961	-------------------V
U.CD.VI1126	-------------------V
CONSENSUS_CPZ	--r--v------V----c-V
CPZ.CD.CPZANT	-------M----V--x---V
CPZ.GA.CPZGAB	V-R--V------V----C-V
CPZ.US.CPZUS	--R--VV-----V-K--C-V

HQMKDCTERQANFLGKIWPS

QUERY	HQMKDCTERQANFLGKIWPS
CONSENSUS_A	-------...ERQANFLGki
A.KE.Q23-CXC-CG	-------...ERQANFLGKI
A.SE.SE6594	-------...ERQANFLGKI
A.SE.SE7253	-------...ERQANFLGKM
A.SE.SE7535	-------...ERQANFLGRI
A.SE.SE8131	-------...ERQANFLGKI
A.SE.SE8538	-------...ERQANFLGKI
A.SE.SE8891	-------...ERQANFLGKI
A.UG.92UG037	-------...ERQANFLGKI
A.UG.U455	-------...ERQANFLGKI
CONSENSUS_B	-------??.eRQAnFLGkI
B.AU.AF128998	-------...ERQANFLGKI
B.-.NL43E9	-------...ERQANFLGKI
B.AU.MBC18	-------...ERQANFLGKI
B.AU.MBC200	-------...ERQANFLGKI
B.AU.MBC925	-------...ERQANFLGKI
B.AU.MBCC54	-H-T---...DRQANFLGKI
B.AU.MBCC98	---I---...ERQANFLGKI
B.AU.MBCD36	-------...ERQANFLGKI
B.CN.RL42	-L------...ERQANFLGKI
B.DE.D31	-------...ERQANFLGKI
B.DE.HAN	-------...ERQANFLGKI
B.ES.89SP061	-------...ERQANFLGKI
B.FR.HXB2	-------...ERQANFLGKI
B.GA.OYI	-------...ERQANFLGKI
B.GB.CAM1	------N...ERQANFLGKI
B.GB.MANC	-------...ERQANFLGKI
B.JP.JH31	------N...ERQANFLGKI
B.NL.3202A21	-------...ERQANFLGKI
B.TW.LM49	-------...ERQANFLGKI
B.US.85WCIPR54	-------...ERQANFLGKI
B.US.AD8	-------...ERQANFLGKI
B.US.BC	-------...ERQANFLGKI
B.US.DH123	-------...ERQANFLGKI
B.US.JRCSF	----E---...ERQANFLGKI
B.US.JRFL	-------...ERQANFLGKI
B.US.MNCG	-------...ERQANFLGKI
B.US.NC7	------I...ERQANFLGKI
B.US.NY5CG	-------...ERQANFLGKI
B.US.P896	-------...ERQANFLGKI
B.US.RF	-------NE.GRQANFLGKI
B.US.SF2	-------...ERQANFLGKI
B.US.WC001	-------...ERQANFLGKI
B.US.WEAU160	-------...ERQANFLGKI
B.US.WR27	---x-xx...ERQAxFLGxI
B.US.YU2	-------...ERQANFLGKI
CONSENSUS_C	-------...ErqAnFLGki
C.BR.92BR025	--V----...ERQANFLGKI
C.BW.96BW01B22	-------...ERQANFLGKI
C.BW.96BW0402	-------...ERQANFLGKI
C.BW.96BW0502	-------...ERQANFLGKI
C.BW.96BW1104	-------...ERRANFLGKI
C.BW.96BW1210	------S...EGQANFLGKI
C.BW.96BW15B03	-------...ERQANFLGKI
C.BW.96BW1626	-------...ERQADFLGKI
C.BW.96BW17A09	----E--...ERQANFLGKI
C.ET.ETH2220	-------...ERQANFLGRL
C.IN.93IN904	-------...ERQANFLGKI
C.IN.93IN905	-------...ERQANFLGKI
C.IN.93IN999	-------...ERQANFLGKI
C.IN.94IN11246	-------...ERQANFLGKI
C.IN.95IN21068	-------...ERQANFLGKI
CONSENSUS_D	-------...ERQANFLGkI
D.CD.84ZR085	-------...ERQANFLGKI
D.CD.ELI	--L----...ERQANFLGRI
D.CD.NDK	-------...ERQANFLGKI
D.CD.Z2Z6	--L----...ERQANFLGKI
D.UG.94UG1141	-------...ERQANFLGKI
CONSENSUS_F	-------...ErQANFLGKI
F.BR.BZ162	-------...EGQANFLGKI
F.CD.VI174	-------...ERQANFLGKI
F.RW.VI69	-------...ERQANFLGKI
CONSENSUS_F1	-------...ERQANFLGKI
F1.BE.VI850	-------...ERQANFLGKI
F1.BR.93BR020.1	-------...ERQANFLGKI
F1.FI.FIN9363	-------...ERQANFLGKI
F1.FR.MP411	-------...ERQANFLGKI
CONSENSUS_F2	-------...ERQANFLGK?
F2.CM.MP255	-------...ERQANFLGKI
F2.CM.MP257	-------...ERQANFLGKM
CONSENSUS_G	----x--...ERQANFLGKI
G.BE.DRCBL	----E--...ERQANFLGKI
G.FI.HH8793	-------...ERQANFLGKI
G.NG.92NG083	----E--...ERQANFLGKI
G.SE.SE6165	-------...ERQANFLGKI
CONSENSUS_H	-------...ERQANFLGKI
H.BE.VI991	-------...GRQANFLGKI
H.BE.VI997	-------...ERQANFLGKI
H.CF.90CF056	-------...ERQANFLGKI
CONSENSUS_J	-------...ERQANFLGKI
J.SE.SE9173	-------...ERQANFLGKI
J.SE.SE9280	-------...ERQANFLGKI
CONSENSUS_K	-------??.eRQANFLGKi
K.BE.VI325	-------...ERQANFLGKI
K.CD.EQTB11C	------S...ERQANFLGKF
K.CM.MP535	-------...ERQANFLGKI
N.CM.YBF30	------KNE.GRQANFLGK-
CONSENSUS_O	------?N..G?QANFLGKY
O.CM.ANT70C	------RN..GKQANFLGKY
O.CM.MVP5180	------KN..GRQANFLGKY
CRF01-AE.CF.90CF40	-------...ERQANFLGKI
CRF01-AE.TH.93TH25	-------...ERQANFLGKI
CRF01-AE.TH.CM240	-------...ERQANFLGKI
CRF01-AE.TH.TH022	-------...ERQANFLGKI
CRF01-AE.TH.TH047	-------...ERQANFLGKI
CRF02_AG.FR.DJ263	-------...EGQANFLGKI
CRF02_AG.FR.DJ264	-------...ERQANFLGKI
CRF02_AG.NG.IBNG	-------...ERQANFLGKI
CRF03_AB.RU.KAL15	-------...ERQANFLGRI
CRF04_cpx.CY.94CY0	-------...ERQANFLGRM
CRF04_cpx.GR.97PVC	-------...ERQANFLGRM
CRF04_cpx.GR.97PVM	------P...ERQANSLGRM
AC.ET.E3099G	-------...ERQANFLGKI
AC.IN.21301	-------...ERQANFLGKI
AC.RW.92RW009	-------...ERQANFLGKI
AC.SE.SE9488	-------...ERQANFLGKI
AC.ZM.ZAM174-21	-------...ERQANFLGKI
AC.ZM.ZAM184	-------...ERQANFLGKI
AC.ZM.ZAM716-17	-------...ERQANFLGKI
ACD.SE.SE8603	-------...ERQANFLGKI
AD.SE.SE6954	-------...ERQANFLGKI
AD.SE.SE7108	-------...ERQANFLGKI
ADHU.NO.NOGIL3	-------...ERQANFLGKI
ADU.CD.MAL	-------...ERQANFLGKI
AG.NG.G3	-------...ERQANFLGKI
AG.SE.SE7812	-------...ERQANFLGKI
AGHU.GA.VI354	-------...ERQANFLGKI
AGJ.AU.BFP90	-------...ERQANFLGKI
AGJ.ML.95ML8	-------...ERQANFLGRI
AGU.CD.Z321	------....ERQANFLGKI
BF.BR.93BR029.4	-------...ERQANFLGKI
DF.CD.VI961	------I...EGQANFLGRV
U.CD.VI1126	-------...ERQANFLGKI
CONSENSUS_CPZ	-------a?n?rqvNFLGK?
CPZ.CD.CPZANT	--L-N-PATNTGKVNFLGKP
CPZ.GA.CPZGAB	-------...GRQVNFLGKG
CPZ.US.CPZUS	-------AGN.RQANFLGKH

ANFLGKIWPSYKGRPGNFLQ

QUERY	ANFLGKIWPSYKGRPGNFLQ
CONSENSUS_A	----------s-----.RPG
A.KE.Q23-CXC-CG	----------R-----.RPG
A.SE.SE6594	----------S-----.RPG
A.SE.SE7253	------M---S-----.RPG
A.SE.SE7535	-----R----S-----.RPG
A.SE.SE8131	----------H-----.RPG
A.SE.SE8538	----------S-----.RPG
A.SE.SE8891	----------NR----.RPG
A.UG.92UG037	----------S-----.RPG
A.UG.U455	----------N-----.RPG
CONSENSUS_B	----------h-----.Rpg
B.AU.AF128998	----------H-----.RPG
B.-.NL43E9	----------H-----.RPG
B.AU.MBC18	----------H-----.RPG
B.AU.MBC200	--------------....RPG
B.AU.MBC925	----------H-----.RPG
B.AU.MBCC54	--------------....RPG
B.AU.MBCC98	----------H-----.RPG
B.AU.MBCD36	----------H-----.RQE
B.CN.RL42	----------H-----.RPG
B.DE.D31	--------------....RPG
B.DE.HAN	----------H-----.RPG
B.ES.89SP061	----------HR----.RPG
B.FR.HXB2	--------------....RPG
B.GA.OYI	----------H-----.RPG
B.GB.CAM1	----------H-----.RPG
B.GB.MANC	----------H-----.RPG
B.JP.JH31	----------S-----.RPG
B.NL.3202A21	----------H-----.RPG
B.TW.LM49	----------H-E---.RPG
B.US.85WCIPR54	----------H-----.RPG
B.US.AD8	----------H-----.RPG
B.US.BC	----------H-----.RPG
B.US.DH123	----------H-E---.RPG
B.US.JRCSF	--------------....RPG
B.US.JRFL	--------------....RPG
B.US.MNCG	----------C-----.R.R
B.US.NC7	----------H-----.RPG
B.US.NY5CG	----------H-----.RPG
B.US.P896	----------H-----.RPG
B.US.RF	----------H-----.RPG
B.US.SF2	--------------....RPG
B.US.WC001	--------------....RPG
B.US.WEAU160	--------S-Q-----.RPG
B.US.WR27	-x---x-R--Hx----.RPG
B.US.YU2	----------H-----.RPG
CONSENSUS_C	----------h----.?RPG
C.BR.92BR025	----------HR----.RPG
C.BW.96BW01B22	----------H-----.RPG
C.BW.96BW0402	----------H-----.RPG
C.BW.96BW0502	----------H-----.RPG
C.BW.96BW1104	----------H-----.RPG
C.BW.96BW1210	----------H-----.RPG
C.BW.96BW15B03	----------H-----.RPG
C.BW.96BW1626	-D--------H-----.RPG
C.BW.96BW17A09	----------H-----.GRPG
C.ET.ETH2220	-----RL---N-----.RPG
C.IN.93IN904	----------H-----.RPG
C.IN.93IN905	----------H-----.RPG
C.IN.93IN999	----------H-----.RPG
C.IN.94IN11246	----------H-----.RPG
C.IN.95IN21068	----------H-----.RPG
CONSENSUS_D	----------H-----.RPG
D.CD.84ZR085	----------H-----.RPG
D.CD.ELI	-----R----H-----.RPG
D.CD.NDK	----------H-----.RPG
D.CD.Z2Z6	----------H-----.RPG
D.UG.94UG1141	----------HN----.RPG
CONSENSUS_F	----------n-----.RPG
F.BR.BZ162	----------H-----.RPG
F.CD.VI174	----------N-----.RPG
F.RW.VI69	----------N-----.RPG
CONSENSUS_F1	----------N-----.RPG
F1.BE.VI850	----------N-----.RPG
F1.BR.93BR020.1	----------N-----.RPG
F1.FI.FIN9363	----------N-----.RPG
F1.FR.MP411	----------N-----.RPG
CONSENSUS_F2	------?---N?----.RPG
F2.CM.MP255	----------NR----.RPG
F2.CM.MP257	------M---N-----.RPG
CONSENSUS_G	----------N-----.RPG
G.BE.DRCBL	----------N-----.RPG
G.FI.HH8793	----------N-----.RPG
G.NG.92NG083	----------N-----.RPG
G.SE.SE6165	----------N-----.RPG
CONSENSUS_H	----------S-----.RPG
H.BE.VI991	----------S-----.RPG
H.BE.VI997	----------S-----.RPG
H.CF.90CF056	----------S-----.RPG
CONSENSUS_J	----------S-----.RPG
J.SE.SE9173	----------S-----.RPG
J.SE.SE9280	----------S-----.RPG
CONSENSUS_K	----------n-----.rPG
K.BE.VI325	----------N-----.KPG
K.CD.EQTB11C	------F--LN-E----.RPG
K.CM.MP535	----------H-----.RPG
N.CM.YBF30	------S-SPF-----.RPG
CONSENSUS_O	------Y--..P-GT..RPG
O.CM.ANT70C	------Y--..P-GT..RPG
O.CM.MVP5180	------Y--..P-GT..RPG
CRF01-AE.CF.90CF40	----------S-----.RPG
CRF01-AE.TH.93TH25	----------N-----.RPG
CRF01-AE.TH.CM240	----------N-----.RPG
CRF01-AE.TH.TH022	----------N-----.RPG
CRF01-AE.TH.TH047	----------N-----.RPG
CRF02_AG.FR.DJ263	----------S-----.RPG
CRF02_AG.FR.DJ264	----------S-----.RPG
CRF02_AG.NG.IBNG	----------S-----.RPG
CRF03_AB.RU.KAL15	-----R----S-----.RPG
CRF04_cpx.CY.94CY0	-----RM---S-----.RPG
CRF04_cpx.GR.97PVC	-----RM---S-----.RPG
CRF04_cpx.GR.97PVM	--S--RM---S-----.RPG
AC.ET.E3099G	----------N-----.RPG
AC.IN.21301	----------H-----.RPG
AC.RW.92RW009	----------N-----.RPG
AC.SE.SE9488	----------S-----.RPG
AC.ZM.ZAM174-21	----------H-----.RPG
AC.ZM.ZAM184	----------H-----.RPG
AC.ZM.ZAM716-17	----------H-----.RPG
ACD.SE.SE8603	----------H-----.RPG
AD.SE.SE6954	----------S-----.RPG
AD.SE.SE7108	----------S-----.RPG
ADHU.NO.NOGIL3	----------S-----.RPG
ADU.CD.MAL	----------H-----.RPG
AG.NG.G3	----------N-----.RPG
AG.SE.SE7812	----------S-----.RPG
AGHU.GA.VI354	----------N-----.RPG
AGJ.AU.BFP90	----------N-----.RPG
AGJ.ML.95ML8	-----R----S-----.RPG
AGU.CD.Z321	----------N-----.RPG
BF.BR.93BR029.4	----------H-----.RPG
DF.CD.VI961	-----RV-L-H-----.RPG
U.CD.VI1126	----------N-----.RPG
CONSENSUS_CPZ	v-----?--??w-????RPG
CPZ.CD.CPZANT	V-----PT-TWW-C...RPG
CPZ.GA.CPZGAB	V-----G---RS-----.RPG
CPZ.US.CPZUS	------H-SPSWSGGSKRPG

Study Subject ID:03RCH89
Study Subject Clone:
Study Subject HLA:A2,A23,B62,B49,Cw10,Cw7
Sequence: Known reactive 20Mer0: IYKRWIILGLNKIVRMYSPT p24(129–148)

Possible HLA

A2	A2.1,A*0201,A*0202,A*0203,A*0204,A*0205,A*0206,A*0207,A*0208,A*0209,A*0210,A*0211,A*0212,A*0213,A*0214,A*0216,A*0217,A*0218,A*0220,A*0
A23	A*2301
B49	B*4901
B62	Bw62,B*1501,B*1504,B*1505,B*1506,B*1507,B*1515,B*1520,B*1524,B*1525,B*1527,B*1530,B*1532,B*1545,B*1548
Cw10	Cw*0302,Cw*0304
Cw7	Cw*0701,Cw*0702,Cw*0704,Cw*0706

Possible Epitopes based on anchor residues

(7-14)	ILGLNKIV	A*0201
(7-14)	ILGLNKIV	A*0202
(7-14)	ILGLNKIV	A*0214
(9-17)	GLNKIVRMY	B*1501
(2-10)	YKRWIILGL	Cw*0702
(9-17)	GLNKIVRMY	Cw*0702
(1-8)	IYKRWIIL	Cw*0702
(3-10)	KRWIILGL	Cw*0702
(10-17)	LNKIVRMY	Cw*0702
(1-10)	IYKRWIILGL	Cw*0702
(8-17)	LGLNKIVRMY	Cw*0702

Anchor Residues Searched

A*0201	X[LM]XXXXXX[VL]
A*0201	X[LM]XXXXX[VL]
A*0201	X[LM]XXXXXXX[VL]
A*0202	X[L]XXXXXX[LV]
A*0202	X[L]XXXXX[LV]
A*0202	X[L]XXXXXXX[LV]
A*0204	X[L]XXXXXX[L]
A*0204	X[L]XXXXX[L]
A*0204	X[L]XXXXXXX[L]
A*0205	X[VLIMQ]XXXXXX[L]
A*0205	X[VLIMQ]XXXXX[L]
A*0205	X[VLIMQ]XXXXXXX[L]
A*0206	X[V]XXXXXX[V]
A*0206	X[V]XXXXX[V]

A*0206	X[V]XXXXXXX[V]
A*0207	X[L][D]XXXXX[L]
A*0207	X[L][D]XXXX[L]
A*0207	X[L][D]XXXXXX[L]
A*0214	X[VQL]XXXXXX[LV]
A*0214	X[VQL]XXXXX[LV]
A*0214	X[VQL]XXXXXXX[LV]
B*1501	X[QL]XXXXXX[FY]
B*1501	X[QL]XXXXX[FY]
B*1501	X[QL]XXXXXXX[FY]
Cw*0304	X[A]XXXXXX[LM]
Cw*0304	X[A]XXXXX[LM]
Cw*0304	X[A]XXXXXXX[LM]
Cw*0702	XXXXXXXX[YFL]
Cw*0702	XXXXXXX[YFL]
Cw*0702	XXXXXXXXX[YFL]

Study Subject ID:03RCH89
Study Subject Clone:
Study Subject HLA:A2,A23,B62,B49,Cw10,Cw7
Sequence: Known reactive 20Mer1: HQMKDCTERQANFLGKIWPS p2p7p1p6(58–77)

Possible HLA

A2	A2.1,A*0201,A*0202,A*0203,A*0204,A*0205,A*0206,A*0207,A*0208,A*0209,A*0210,A*0211,A*0212,A*0213,A*0214,A*0216,A*0217,A*0218,A*0220,A*0
A23	A*2301
B49	B*4901
B62	Bw62,B*1501,B*1504,B*1505,B*1506,B*1507,B*1515,B*1520,B*1524,B*1525,B*1527,B*1530,B*1532,B*1545,B*1548
Cw10	Cw*0302,Cw*0304
Cw7	Cw*0701,Cw*0702,Cw*0704,Cw*0706

Possible Epitopes based on anchor residues

(5-13)	DCTERQANF	Cw*0702
(6-14)	CTERQANFL	Cw*0702
(6-13)	CTERQANF	Cw*0702
(7-14)	TERQANFL	Cw*0702
(4-13)	KDCTERQANF	Cw*0702
(5-14)	DCTERQANFL	Cw*0702

Anchor Residues Searched

A*0201	X[LM]XXXXXX[VL]
A*0201	X[LM]XXXXX[VL]
A*0201	X[LM]XXXXXXX[VL]
A*0202	X[L]XXXXXX[LV]
A*0202	X[L]XXXXX[LV]
A*0202	X[L]XXXXXXX[LV]
A*0204	X[L]XXXXXX[L]
A*0204	X[L]XXXXX[L]
A*0204	X[L]XXXXXXX[L]
A*0205	X[VLIMQ]XXXXXX[L]
A*0205	X[VLIMQ]XXXXX[L]
A*0205	X[VLIMQ]XXXXXXX[L]
A*0206	X[V]XXXXXX[V]
A*0206	X[V]XXXXX[V]
A*0206	X[V]XXXXXXX[V]
A*0207	X[L][D]XXXXX[L]
A*0207	X[L][D]XXXX[L]
A*0207	X[L][D]XXXXXX[L]
A*0214	X[VQL]XXXXXX[LV]

A*0214	X[VQL]XXXXX[LV]
A*0214	X[VQL]XXXXXXX[LV]
B*1501	X[QL]XXXXXX[FY]
B*1501	X[QL]XXXXX[FY]
B*1501	X[QL]XXXXXXX[FY]
Cw*0304	X[A]XXXXXX[LM]
Cw*0304	X[A]XXXXX[LM]
Cw*0304	X[A]XXXXXXX[LM]
Cw*0702	XXXXXXXX[YFL]
Cw*0702	XXXXXXX[YFL]
Cw*0702	XXXXXXXXX[YFL]

Study Subject ID:03RCH89
Study Subject Clone:
Study Subject HLA:A2,A23,B62,B49,Cw10,Cw7
Sequence: Known reactive 20Mer2: ANFLGKIWPSYKGRPGNFLQ p2p7p1p6(68–87)

Possible HLA

A2	A2.1,A*0201,A*0202,A*0203,A*0204,A*0205,A*0206,A*0207,A*0208,A*0209,A*0210,A*0211,A*0212,A*0213,A*0214,A*0216,A*0217,A*0218,A*0220,A*0
A23	A*2301
B49	B*4901
B62	Bw62,B*1501,B*1504,B*1505,B*1506,B*1507,B*1515,B*1520,B*1524,B*1525,B*1527,B*1530,B*1532,B*1545,B*1548
Cw10	Cw*0302,Cw*0304
Cw7	Cw*0701,Cw*0702,Cw*0704,Cw*0706

Possible Epitopes based on anchor residues

(3-11)	FLGKIWPSY	B*1501
(3-11)	FLGKIWPSY	Cw*0702
(10-18)	SYKGRPGNF	Cw*0702
(11-19)	YKGRPGNFL	Cw*0702
(4-11)	LGKIWPSY	Cw*0702
(11-18)	YKGRPGNF	Cw*0702
(12-19)	KGRPGNFL	Cw*0702
(2-11)	NFLGKIWPSY	Cw*0702
(9-18)	PSYKGRPGNF	Cw*0702
(10-19)	SYKGRPGNFL	Cw*0702

Anchor Residues Searched

A*0201	X[LM]XXXXXX[VL]
A*0201	X[LM]XXXXX[VL]
A*0201	X[LM]XXXXXXX[VL]
A*0202	X[L]XXXXXX[LV]
A*0202	X[L]XXXXX[LV]
A*0202	X[L]XXXXXXX[LV]
A*0204	X[L]XXXXXX[L]
A*0204	X[L]XXXXX[L]
A*0204	X[L]XXXXXXX[L]
A*0205	X[VLIMQ]XXXXXX[L]
A*0205	X[VLIMQ]XXXXX[L]
A*0205	X[VLIMQ]XXXXXXX[L]
A*0206	X[V]XXXXXX[V]
A*0206	X[V]XXXXX[V]
A*0206	X[V]XXXXXXX[V]

A*0207	X[L][D]XXXXX[L]
A*0207	X[L][D]XXXX[L]
A*0207	X[L][D]XXXXXX[L]
A*0214	X[VQL]XXXXXX[LV]
A*0214	X[VQL]XXXXX[LV]
A*0214	X[VQL]XXXXXXX[LV]
B*1501	X[QL]XXXXXX[FY]
B*1501	X[QL]XXXXX[FY]
B*1501	X[QL]XXXXXXX[FY]
Cw*0304	X[A]XXXXXX[LM]
Cw*0304	X[A]XXXXX[LM]
Cw*0304	X[A]XXXXXXX[LM]
Cw*0702	XXXXXXXX[YFL]
Cw*0702	XXXXXXX[YFL]
Cw*0702	XXXXXXXXX[YFL]

This table lists epitopes that are experimentally observed to be presented by a HLA type carried by the patient, but the de£ned epitope has substitutions relative to the peptides from your reference strains and so might be missed by your reagents: in HXB2 for Gag, Pol; MN for Env; BRU for Nef, relative to most B clade Sequences in the database:

Protein	Epitope in Database	Epitope in Ref. strain	Epitope in Consensus B	HLA	Notes
p17(77–85)	SLFNTVATL	SLYNTVATL	SLYNTVATL	A*0201	
RT(179–187)	VIYQYMMDL	VIYQYMDDL	VIYQYMDDL	A2	
RT(179–187)	VIYQYMMDL	VIYQYMDDL	VIYQYMDDL	A2, A*0202	
RT(308–317)	EILKEPVGHV	EILKEPVHGV	EILKEPVHGV	A*0201	
gp160(121–129)	KLTPLCVSL	KLTPLCVTL	KLTPLCVTL	A2	
gp160(192–200)	KLTSCNTSV	RLISCNTSV	RLISCNTSV	A2	
gp160(192–200)	TLTSCNTSV	RLISCNTSV	RLISCNTSV	A2	
gp160(192–200)	TLTSCNTSV	RLISCNTSV	RLISCNTSV	A2.1	
gp160(311–320)	RGPGRAFVTI	IGPGRAFYTT	IGPGRAFYTT	A*0201	
gp160(311–320)	RGPGRAFVTI	IGPGRAFYTT	IGPGRAFYTT	A2	
gp160(311–320)	MGPKRAFYAT	IGPGRAFYTT	IGPGRAFYTT	A2	
gp160(369–375)	PEIVTHS	PEIVMHS	PEIVMHS	A2	
gp160(377–387)	NSGGEFFYSNS	NCGGEFFYCNT	NCGGEFFYCNT	A2	
gp160(700–708)	AVLSVVNRV	AVLSIVNRV	AVLSIVNRV	A2	
gp160(747–755)	RLVNGSLAL	RLVHGFLAI	RLVDGFLAL	A2	
gp160(770–778)	RLRDLLLIV	HHRDLLLIA	RLRDLLLIV	A*0201	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A*0201	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A2	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A2.1	
gp160(814–822)	LLNATDIAV	LLNATAIAV	LLNATAIAV	A2	
Nef(135–143)	YLPTFGWCY	YPLTFGWCY	YPLTFGWCF	B49	
Nef(136–145)	PLTFGWCFKL	PLTFGWCYKL	PLTFGWCFKL	A2	

Table 1: **p17**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85 Clade A)	SLFNTVATL	HIV-1 infection	human(A*0201)	[Dorrell (1999)]
	• Epitope SL9: CTL responses in three individuals with non-clade B infections were studied, 2 with subtype A infections, 1 with subtype C – their infections all originated in East Africa • This epitope is most commonly SLYNTVATL in B subtype, and CTL from the C subtype infection did not recognize B clade gag or the 3Y form of the epitope, but do recognize the predominant A and C clade form, SLFNTVATL				

Table 2: **RT**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(179–187)	RT()	VIYQYMMDL	HIV-1 exposure	human(A2)	[Rowland-Jones (1998a)]
	• A CTL response was found in exposed but uninfected prostitutes from Nairobi using previously-de£ned B clade epitopes that tended to be conserved in A and D clades – such cross-reactivity could protect against both A and D and confer protection in Nairobi where both subtypes are circulating • The A and D consensus sequences are both VIYQYMMDL				
RT(179–187)	Pol()	VIYQYMMDL	HIV-1 exposure	human(A2, A*0202)	[Rowland-Jones (1998b)]
	• HIV-speci£c CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes • This epitope is conserved among A, B and D clade viruses				
RT(308–317)	RT()	EILKEPVGHV	HIV-1 infection	human(A*0201)	[van der Burg (1997), Menendez-Arias (1998)]
	• Recognized by CTL from a long-term survivor, SPIETVPVKL was also recognized • Recognized by CTL from a progressor, EELRQHLLRW and TWETWWTEYW were also recognized				

Table 3: **gp160**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(121–129)	gp120(121–129)	KLTPLCVSL	*in vitro* stimulation	human(A2)	[Zarling (1999)]
	• This study compares the ability of macrophages and dendritic cells to stimulate primary responses in CD8+ lymphocytes isolated from HLA-appropriate HIV-uninfected donors using peptide-pulsed APC – the dendritic cells performed better as APC for the stimulation of primary responses • Strong CTL responses were elicited by the epitopes DRFYKTLRA and GEIYKRWII when presented by either immature or mature dendritic cells – macrophages were not able to prime a CTL response against DRFYKTLRA • A weak response to KLTPLCVSL was stimulated using macrophages as the APC • No detectable response was observed for the following previously-de£ned HIV epitopes: KIRLRPGGK, ILKEPVHGV, IRLRPGGK, GPKVKQWPL				
gp160(192–200)	gp120(192–199 HXB2R)	KLTSCNTSV	HIV-1 infection	human(A2)	[Brander (1995)]
	• Epitope predicted on HLA binding motif, and studied in the context of inclusion in a synthetic vaccine				
gp160(192–200)	gp120(197–205)	TLTSCNTSV	no CTL shown	human(A2)	[Garboczi (1992)]
	• Crystallization of HLA-A2 molecules complexed with antigenic peptides – refers to Dadaglio *et al* 1991				
gp160(192–200)	gp120(199–207)	TLTSCNTSV	peptide immunization and HIV-1 infection	human(A2.1)	[Brander (1996)]
	• This epitope was recognized by PBMC from 6/14 HIV+ asymptomatic patients • This epitope was used along with pol CTL epitope ALQDSGLEV and a tetanus toxin T helper epitope for a synthetic vaccine • This vaccine failed to induce a CTL response, although a helper response was evident				
gp160(311–320)	gp160(318–327 IIIB)	RGPGRAFVTI	CTL line from HIV-donor	human(A*0201)	[Alexander-Miller (1996)]
	• This immunogenic peptide does not have the known binding motif for A2.1 • The same optimal peptide for this human HLA-A2.1 epitope was observed for a murine H-2 D^d epitope				
gp160(311–320)	gp160(318–327 IIIB)	RGPGRAFVTI	vaccinia IIIB gp160	human(A2)	[Achour (1996)]
	• Individual was immunized with rec vaccinia gp160 IIIB and boosted with puri£ed gp160 • Lysis only occurs with IIIB P18 peptide pulsed onto autologous targets; MN, RF, SIMI P18 peptides fail to stimulate CTL • Restimulating immune cells from gp160 IIIB vaccinees with MN, RF, or SIMI P18 did not enhance the MN, RF, or SIMI speci£c CTL response				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(311–320)	gp160(318–327 SIMI)	MGPKRAFYAT	vaccinia SIMI gp160	human(A2)	[Achour (1996)]
	• Individual was immunized with rec vaccinia gp160 SIMI and boosted with puri£ed recombinant gp160 SIMI • P18 MN and RF peptides were able to stimulate the HIV-speci£c CTL that arose in response to the SIMI vaccination, thus the P18 MN peptide (IGPGRAFYTT) and the P18 RF peptide (KGPGRVIYAT) could cross-react • The P18 IIIB peptide does not cross-react (RGPGRAFVTI in the epitope region) • gp160 SIMI primed immune cells could generate a signi£cantly broader speci£city when stimulated with P18 MN or P18RF peptides, but not P18 IIIB				
gp160(369–375)	gp120(374–380 BRU)	PEIVTHS	HIV-1 infection	human(A2)	[Dadaglio (1991)]
	• De£ned through blocking CTL activity, and Env deletions				
gp160(377–387)	gp120(377–387)	NSGGEFFYSNS		human(A2)	[Hickling (1990)]
	• Peptides recognized by class I restricted CTL can bind to class II				
gp160(700–708)	gp41(705–714)	AVLSVVNRV	HIV-1 infection	human(A2)	[Ferris (1999)]
	• This epitope is processed by a TAP1/2 dependent mechanism				
gp160(747–755)	gp41(747–755)	RLVNGSLAL	HIV-1 infection	human(A2)	[Parker (1992)]
	• Studied in the context of HLA-A2 peptide binding				
gp160(770–778)	Env(679–777)	RLRDLLLIV	HIV-1 infection	human(A*0201)	[Kmieciak (1998)]
	• CTL responses in six patients to four Env epitopes were studied: D2: LLNATAIAV, 5.3: RLRDLLLIV, D1: KLTPLCVTL, and 4.3: QMHEDIISL – all have A2 anchor residues • The C terminal epitopes (D2 and 5.3) were highly variable and the variability was considered responsible for limited CTL response, while D1 and 4.3, N-terminal epitopes, were much more conserved and gave evidence of high levels of CTL response *in vitro* • Peptides 5.3 and D2 bound to HLA A*0201 with low af£nity and were variable, particularly D2;				
gp160(813–822)	gp41(814–823 LAI)	SLLNATDIAV	MN rec gp160	human(A*0201)	[Dupuis (1995)]
	• Of two CTL clones, one reacted only with 815-823, the other with 814-823 and 815-823 • Noted to be A*0201 in Brander *et al*., 1999 database				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(813–822)	gp41(814–823)	SLLNATDIAV	HIV-1 infection	human(A2)	[Kundu (1998b)]
	• Allogeneic dendritic cells (DCs) were obtained from HLA-identical siblings, pulsed with rgp160 MN or A2-restricted HIV-1 epitope peptides, and infused monthly into six HIV-infected patients • 1/6 showed increased env-speci£c CTL and increased lymphoproliferative responses, 2/6 showed increase only in proliferative responses, and 3/6 showed no change – pulsed DCs were well tolerated • SLLNATDIAV is a conserved HLA-A2 epitope included in this study – 4/6 patients had this sequence as their HIV direct sequence, and 3 of these had a detectable CTL response – the other two had either the sequence SLFNAIDIAV or SLLNTTDIVV and no detectable CTL response • CTL demonstrated against peptide-coated target, epitope is naturally processed and enhancible with vaccine				
gp160(813–822)	Env(814–823 Clade B)	SLLNATDIAV	HIV-1 MN rgp160	human(A2.1)	[Kundu (1998a)]
	• Ten HIV-1+ HLA A2 asymptomatic individuals were given two courses of HIV-1 MN rgp160 vaccine over a 2 year period • Two hundred and £fty three HIV-1 peptides of 9 or 10 aa possessing the HLA-A2.1 binding motif (Leu at position 2, Val at the C terminus) were identi£ed in gp160, of which 25 had a high or intermediate binding af£nity • Eleven peptides were studied that had high HLA-A2 binding af£nity – a CTL response was detected to 9/11 peptides in at least 1 individual • CTL responses after reimmunization may include recall responses – only individuals with vaccine cross-reactive sequences prior to vaccination showed detectable CTL responses • CTL to overlapping peptides in this region gave a positive response in the greatest number of patients • ALTERNATIVE EPITOPES: LLNATDIAV and LLNATDIAVA – CTL were induced by vaccine in those that had the sequence SLLNATAIAVA in their own infection, but not in those with: NLLNTIAIAVA or NLFNTTAIAVA or SLLNATAITVA				
gp160(814–822)	gp41(815–823 LAI)	LLNATDIAV	MN rec gp160	human(A2)	[Dupuis (1995)]
	• Of two CTL clones, one reacted only with 815-823, the other with 814-823 and 815-823				

Table 4: **Nef**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(135–143)	Nef()	YLPTFGWCY	HIV-1 exposure	human(B49)	[Rowland-Jones (1998b)]
	• HIV-speci£c CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes • This epitope is conserved among A and B clade viruses • The Clade D version of the epitope, YPLTFGWCF, was preferentially recognized by CTL				
Nef(136–145)	Nef(136–145)	PLTFGWCFKL	HIV-1 infection	human(A2)	[Durali (1998)]
	• Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • Patient B18 had the greatest breadth and diversity of response, and recognized Gag SLYNTVATL and Nef PLTFGWCFKL				

Table 5: **All De£ned Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(129–136)	p24(263–270 SF2)	IYKRWIIL	HIV-1 infection	human(A*2402)	[Ikeda-Moore (1997)]
	• De£ned using reverse immunogenetics – 59 HLA-A*2402 binding peptides were predicted by searching for A*2402 anchors in HIV proteins (Tyr at 2, and Phe, Leu or Ile at the C term) – 53 of the 59 peptides bound A*2402 • This peptide induced CTL in 1/4 HIV-1+ people tested • IYKRWIIL bound to A*2402 with medium strength, the epitope can be processed in a vaccinia construct and presented – two speci£c CTL clones were obtained				
p24(129–138)	p24(263–272 SF2)	IYKRWIILGL	HIV-1 infection	human(A*2402)	[Ikeda-Moore (1997)]
	• De£ned using reverse immunogenetics – 59 HLA-A*2402 binding peptides were predicted by searching for A*2402 anchors in HIV proteins (Tyr at 2, and Phe, Leu or Ile at the C term) – 53 of the 59 peptides bound A*2402 • This peptide induced CTL in 1/4 HIV-1+ people tested • IYKRWIILGL bound to A*2402 with medium strength, the epitope can be processed in a vaccinia construct and presented – two speci£c CTL clones were obtained				
p24(129–138)	p24(263–272)	IYKRWIILGL	HIV-1 infection	human(B27)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned peptides from this database were used to screen for gamma interferon responses to other epitopes • 1/11 of the A2+ individuals was B27 and responded to IYKRWIILGL				
p24(130–148)	p24(265–280 BRU)	YKRWIILGLNKIVRMYSPT	HIV-1 infection	human(B27)	[Dadaglio (1991)]
	• Used as a positive control for HLA speci£city				
p24(131–139)	p24(263–272)	KRWIILGNK	HIV-1 infection	human(B27)	[Durali (1998)]
	• Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • One of the patients was shown to react to this epitope: KRWIILGNK				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–139)	Gag(265–273)	KRWIILGLN	HIV-1 infection	chimpanzee(Patr-B*03)	[Balla-Jhagjhoorsingh (1999)]
	• Certain HLA-alleles have been associated with long-term survival – among them are HLA-B*27 and HLA-B*57 • Of more than 150 chimpanzees that have been reported to be infected with HIV-1, only one has developed AIDS • CTL responses were studied in two HIV-1 infected chimpanzees that have strong CTL responses, and they were found to respond to highly conserved epitopes that are recognized in humans in the context of HLA-B*27 and HLA-B*57 • The human HLA protein which presents this Patr-B*03 epitope is HLA B*2705 but the amino acid sequences in the binding pockets of HLA-B*2705 and Patr-B*03 are distinctive				
p24(131–140)	Gag(263–272 LAI)	KRWILLGLNK	HIV-1 infection	human()	[Buseyne (1993a)]
	• Vertical transmission of HIV ranges from 13% to 39% • Primary assays showed cytotoxic activity against at least one HIV protein was detected in 70% of infected children • Epitopes recognized in £ve children were mapped using synthetic peptides and secondary cultures • Patient EM28 (CDC P2A) had a CTL response to four epitopes in Gag				
p24(131–140)	p24(263–272)	KRWIILLGLNK	HIV-1 infection	human(B*27)	[Huang (2000)]
	• The single cell ELISPOT assay was optimized and highly speci£c, and found to work well even after the primary cells had been frozen and thawed • Increases in gamma interferon producing cells were observed in response to anti-retroviral therapy using single cell IFN-gamma-production ELISPOT • In 3/3 HLA A*02, B*27 individuals, the dominant response in gag measured by both gamma IFN production and T cell lysis was to the B27 epitope, KRWIILLGLNK, not the A2 SLYNTVATL epitope				
p24(131–140)	p24(263–272 SF2)	KRWIILGLNK	HIV-1 infection	human(B*27)	[McAdam (1998)]
	• Epitope invariant across clades A, B, C, and D				
p24(131–140)	p24(260–269 HIV-2)	RRWIQLGLQK		human(B*2703)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*2703 epitope				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–140)	p24()	KRWIILGGLNK	HIV-1 infection	human(B*2705)	[Wilson (2000)]
	• Three individuals with highly focused HIV-speci£c CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-speci£c CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-speci£c T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • Tetramers with peptide variants KRWIILGGLNK and KRWIIMGGLNK were used – CTL from most B27 donors recognize both variants, although one of the three subjects recognized only KRWIILGGLNK • ELISPOT was used to test a panel of CTL epitopes that had been de£ned earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWIILGGLNK • The subject with A*0201 had a moderatly strong strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL			
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B*2705)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*2705 epitope				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B*2705,B27)	[Goulder (1997b), Goulder (1997a)]
	• HLA-B*2705 is associated with slow HIV disease progression • 11/11 HLA-B*2705 donors make a response to this epitope, usually an immunodominant response • This is a highly conserved epitope • The HLA-B*2705 binding motif includes R at position 2, and L in the C-term position • [Goulder (1997a)] is a review on CTL immune escape that discusses this epitope in the context of the dif£culty in detection of immune escape – KRWIILGLNK and an R2K change, KKWIILGLNK, show little difference in titration curves, yet the K2 variants fail to bind to targets for more than 1 hour, while the R2 form can sensitize lysis by CTL for over 24 hours – minigene transfection experiments con£rmed the importance of this for the CTL response				
p24(131–140)	p24(260–269 HIV-2)	RRWIQLGLQK		human(B27)	[Brander & Walker(1996)]
	• HIV-2, HLA-B*2703, S. Rowland-Jones, Pers. Comm.				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B27)	[Fan (1997)]
	• The capacity of dendritic cells to process and present antigen and stimulate anti-HIV-1 CTL memory responses was studied				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–140)	Gag(263–272)	KRWIILGLNK	HIV-1 infection	human(B27)	[Zheng (1999)]
	• Protein delivery (gp160 LAV, p66 LAV, and p24 NY5) to human dendritic cells (DC) with liposomes provides enhanced memory CTL response relative to delivery of protein alone • Chloroquine administration enhanced epitope presentation, and brefeldin A and peptide aldehyde inhibitors inhibited antigen presentation, suggesting epitopes were processed by classical proteasome pathway • The CTL response to p24 was measured in individuals with a response to B27-KRWIILGLNK				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B27)	[Wilson (1998)]
	• HIV+ individuals were followed longitudinally using MHC tetramers in combination with 14 anti-BV chain MAbs, and clonal expansion of HIV-speci£c T cells was followed *in vivo* • Seven HIV+ people were studied, and all showed expansions of particular TCR BV clones, often several, relative to uninfected controls • Three patients were followed in detail, TCR VB expansions persisted for 2 to 3 years, with occasional transient increases				
p24(131–140)	p24()	KRWIILGLNK	HIV infection	human(B27)	[Rowland-Jones (1997)]
	• Described in this review as the £rst identi£ed HIV CTL epitope				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B27)	[Buseyne (1993b)]
	• Clustering of Gag p24 CTL epitopes recognized in 29 HIV-infected people				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B27)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes				
p24(131–140)	p24(263–272)	KRWIIMGLNK	HIV-1 infection	human(B27)	[Klenerman (1994)]
	• Naturally occurring variant KRWIILGLNK may act as antagonist				
p24(131–140)	p24(263–272)	KRWIIMGLNK	HIV-1 infection	human(B27)	[Klenerman (1995)]
	• Naturally occurring variant KRWIILGLNK may act as antagonist				
p24(131–140)	p24(265–274)	KRWIILGLNK	HIV infection	human(B27)	[Moss (1995)]
	• In one individual, TCR usage changed over time indicating that new populations of CTL can be recruited • TCR usage showed a CTL clonal response to this epitope that persisted over 5 years • CTL clones speci£c for HIV epitopes may represent between 0.2 and 1% of the total CD8+ population of T cells				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–140)	p24(265–276)	KRWIILGLNK		human(B27)	[Carreno (1992)]
	• Included in HLA-B27 binding peptide competition study				
p24(131–140)	p24(265–274 SF2)	KRWIILGLNK	HIV-1 infection	human(B27)	[Phillips (1991), Goulder (1997a)]
	• Longitudinal study of CTL escape mutants – little variation was observed in the immunodominant B27 epitope, relative to B8 epitope • [Goulder (1997a)] is a review of immune escape that points out that there may be a protective effect associated with B27, and that HLA-B8 individuals tend to progress more rapidly than HLA B27 patients				
p24(131–140)	p24(263–272)	KRWIILGLNK	HIV-1 infection	human(B27)	[Nietfeld (1995), Goulder (1997a)]
	• Single point mutations were introduced and viral viability and CTL recognition tested – an Arg to Lys change at anchor position P2 abrogates binding to B27, but doesn„t change viral viability *in vitro* • [Goulder (1997a)] is a review of immune escape that summarizes this study				
p24(131–140)	p24(263–272)	KRWIIMGNK	HIV-1 infection	human(B27)	[Nowak (1995)]
	• Longitudinal study of CTL response and immune escape – the form KRWIILGNK was also found, and both forms stimulate CTL				
p24(131–140)	p24(263–272)	KRWIIMGLNK	HIV-1 infection	human(B27)	[Goulder (1997c), Goulder (1997a)]
	• Six HLA-B27 donors studied make a strong response to this epitope • In 4/6 cases, this was the immunodominant or only CTL response • Two of the cases had an epitope switch to the form KKWIIMGLNK during a period of rapid decline to AIDS, following their asymptomatic period • The arginine to lysine switch is in an anchor residue, and results in immune escape due to severely diminished binding to the B27 molecule • [Goulder (1997a)] is a review of immune escape that summarizes this study in the context of CTL escape to £xation				
p24(131–140)	p24()	KRWIILGLNK		human(B27)	[Rowland-Jones (1999)]
	• CTL responses in seronegative highly HIV-exposed African female sex workers in Gambia and Nairobi were studied – these women had no delta 32 deletion in CCR5 • In Gambia there is exposure to both HIV-1 and HIV-2, CTL responses to B35 epitopes in exposed, uninfected women are cross-reactive, and the B35 allele seems to be protective • HIV-2 sequence: RRWIQLGLQK – this epitope was not HIV-1 and HIV-2 cross-reactive				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–140)	Gag()	KRWILGLNK	none, computer prediction	(B27)	[Schafer (1998)]
	• This study uses EpiMatrix for T cell epitope prediction to identify possible HLA-B27 and A-2 CTL epitopes in HIV • Based on EpiMatrix predictions, 28 peptides were synthesized and tested using T2 binding assays for potential HLA A2 or B27 binding, and 12 of these were shown to bind to the predicted HLA molecule • Two of these 12 peptides had been previously identi£ed as CTL epitopes: HLA-B27 KRWILGLNK and HLA-A2 ILKEPVHGV • This peptide sequence is not conserved between clades, but is found in most B clade isolates				
p24(131–140)	p24(263–282)	KRWIILGLNK	HIV-1 infection	human(B27)	[Bernard (1998)]
	• This study focuses on six rare long-term survivor HIV-infected people who were infected for many years without exhibiting immune dysregulation – such immunologically normal HIV-infected (INHI) cases occur at a frequency between 0.1 and 1% in the infected population • No direct CTL were found in any of the six INHIs, but above background CTLp activity was founded in 3/6 INHIs • Epitope sequences were deduced from larger reactive peptides based on HLA binding motifs – XRXXXXXXXK is a B*2705 binding motif				
p24(131–142)	p24(265–276)	KRWIILGLNKIV	no CTL shown	human(B27)	[Jardetzky (1991)]
	• Epitope examined in the context of peptide binding to HLA-B27				
p24(131–142)	p24(263–274 LAI)	KRWIILGLNKIV	HIV-1 infection	human(B27)	[Fan (1997)]
	• The capacity of dendritic cells to process and present antigen and stimulate anti-HIV-1 CTL memory responses was studied				
p24(131–145)	p24()	KRWILGLNKIVRMY	HIV-1 infection	human()	[Goulder (2000)]
	• The CTL-dominant response was focused on this epitope in a HIV+ African American living in Boston with unknown HLA – this epitope did not fall within the three most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p24(131–145)	p24(263–277 LAI)	KRWIILGLNKIVMRY	HIV-1 infection	human(A33)	[Buseyne (1993b)]
	• Clustering of Gag p24 CTL epitopes recognized in 29 HIV-infected people				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–145)	p24(266–277)	KRWIILGLNKIVRMY	rec gag-vaccinia	human(B27)	[Nixon (1988)]
	• Gag CTL epitope mapped with rec gag-vaccinia and synthetic peptides • This was the £rst HIV-1 epitope to be mapped				
p24(131–145)	p24(266–277 LAI)	KRWIILGLNKIVMRY	HIV-1 infection	human(B27)	[Meyerhans (1991)]
	• Longitudinal study showing persistence of epitope despite CTL activity				
p24(131–145)	p24(265–279)	KRWIILGLNKIVRMY	HIV-1 infection	human(B27)	[Nixon (1990), Rowland-Jones (1999)]
	• HIV-1 and HIV-2 cross-reactive CTL clone, highly conserved epitope • Reviewed in Rowland-Jones99, notes that it did not appear cross-reactive with HIV-2 in Rowland-Jones98, HIV-2 form: RRWIQL-GLQK				
p24(131–146)	p24(265–279)	KRWIILGLNKIVRMYC	HIV-1 infection	human(B27)	[Bouillot (1989)]
	• HLA-B27 restricted epitope also binds to HLA-A2 and HLA-B37 in solid phase assay				
p24(132–145)	Gag()	KWILGLNKIVRMY	HIV-infection	human()	[Weekes (1999a)]
	• Peptide 728: Memory CTL speci£c for HIV-1 may contribute to oligoclonal expansions within the CD57+ CD28- CD8+ CTLp populations				
p24(132–145)	Gag()	KWILGLNKIVRMY	HIV-infection	human(B27)	[Weekes (1999b)]
	• Peptide 728: Almost all CD8+ T cells are CD28+ at birth, and the proportion of CD28-CD8+ cells increases with age – this study examines the contribution of CD8+CD28- cells to CTL memory pools for CTL clones speci£c for two persistent human viruses, CMV and HIV – clones were found to be similarly distributed the CD28 depleted cell population • HIV CTL responses to 3 Env and 2 Gag peptides were studied • The clonal composition of the TCR Vbeta responses were studied and was found to be highly focused, with one TCR beta-chain sequence tending to dominate the peptide-speci£c response – clones to this epitope were Vbeta22.1				
p24(134–143)	p24()	IILGLNKIVR	HIV-1 exposure	human(A33)	[Rowland-Jones (1998b)]
	• HIV-speci£c CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes • This epitope is conserved among A, B and D clade viruses				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(136–145)	p24(268–277 LAI)	LGLNKIVRMY	Predicted from larger peptide	human(Bw62)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes • Also P. Johnson, Pers. Comm.				
p24(136–146)	p24(271–281)	LGLNKIVRMYS	HIV-1 infection	human(B62)	[Lubaki (1997)]
	• Eighty two HIV-1-speci£c CTL clones from 5 long-term non-progressors were isolated and analyzed for breadth of response • A sustained Gag, Env and Nef response was observed, and clones were restricted by multiple HLA epitopes, indicating a polyclonal response • A subject who was B62+ had CTL that recognized this peptide, p17 KIRLRPGGKKKYKL, and one additional unknown epitope • The two clones that recognized this epitope used two different Vβ genes, further demonstrating a polyclonal response				
p24(137–145)	p24()	GLNKIVRMY	HIV-1 infection	human()	[Goulder (2000)]
	• The CTL-dominant response was focused on this epitope in a HIV+ South African living in Durban, HLA A2/- B5802/62 Cw4/6 – this epitope did not fall within the three most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p24(137–145)	p24(272–280 LAI)	GLNKIVRMY	HIV-1 infection	human(B*1501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*1501 epitope				
p24(137–145)	p24(272–280 LAI)	GLNKIVRMY	HIV-1 infection	human(B62)	[Goulder (1997a)]
	• This paper is a review of CTL and immune evasion, but it presents a study of a shift from an HLA-A*0201 response to SLYNTVATL, to a B62 response to GLNKIVRMY • As long as a strong CTL response to SLYNTVATL was evident, the epitope variants SLFNTVATL or SLYNTIATL dominated the viral population – eventually the CTL response to the index peptide became undetectable, the CTL response shifted to a focus on GLNKIVRMY, and the index peptide SLYNTVATL once again established itself as the dominant form				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(137–145)	p24()	GLNKIVRMY	HIV-1 infection	human(B62)	[Goulder (2000)]

- The CTL-dominant response was focused on this epitope in a HIV+ African American living in Boston – this epitope did not fall within the three most recognized peptides in the study
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Table 6: **All De£ned Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(129–136)	p24(263–270 SF2)	IYKRWIIL	HIV-1 infection	human(A*2402)	[Ikeda-Moore (1997)]
	• De£ned using reverse immunogenetics – 59 HLA-A*2402 binding peptides were predicted by searching for A*2402 anchors in HIV proteins (Tyr at 2, and Phe, Leu or Ile at the C term) – 53 of the 59 peptides bound A*2402 • This peptide induced CTL in 1/4 HIV-1+ people tested • IYKRWIIL bound to A*2402 with medium strength, the epitope can be processed in a vaccinia construct and presented – two speci£c CTL clones were obtained				
p24(129–138)	p24(263–272 SF2)	IYKRWIILGL	HIV-1 infection	human(A*2402)	[Ikeda-Moore (1997)]
	• De£ned using reverse immunogenetics – 59 HLA-A*2402 binding peptides were predicted by searching for A*2402 anchors in HIV proteins (Tyr at 2, and Phe, Leu or Ile at the C term) – 53 of the 59 peptides bound A*2402 • This peptide induced CTL in 1/4 HIV-1+ people tested • IYKRWIILGL bound to A*2402 with medium strength, the epitope can be processed in a vaccinia construct and presented – two speci£c CTL clones were obtained				
p24(129–138)	p24(263–272)	IYKRWIILGL	HIV-1 infection	human(B27)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned peptides from this database were used to screen for gamma interferon responses to other epitopes • 1/11 of the A2+ individuals was B27 and responded to IYKRWIILGL				
p24(130–148)	p24(265–280 BRU)	YKRWIILGLNKIVRMYSPT	HIV-1 infection	human(B27)	[Dadaglio (1991)]
	• Used as a positive control for HLA speci£city				
p24(131–139)	p24(263–272)	KRWIILGNK	HIV-1 infection	human(B27)	[Durali (1998)]
	• Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • One of the patients was shown to react to this epitope: KRWIILGNK				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–139)	Gag(265–273)	KRWIILGLN	HIV-1 infection	chimpanzee(Patr-B*03)	[Balla-Jhagjhoorsingh (1999)]
	• Certain HLA-alleles have been associated with long-term survival – among them are HLA-B*27 and HLA-B*57 • Of more than 150 chimpanzees that have been reported to be infected with HIV-1, only one has developed AIDS • CTL responses were studied in two HIV-1 infected chimpanzees that have strong CTL responses, and they were found to respond to highly conserved epitopes that are recognized in humans in the context of HLA-B*27 and HLA-B*57 • The human HLA protein which presents this Patr-B*03 epitope is HLA B*2705 but the amino acid sequences in the binding pockets of HLA-B*2705 and Patr-B*03 are distinctive				
p24(131–140)	Gag(263–272 LAI)	KRWILLGLNK	HIV-1 infection	human()	[Buseyne (1993a)]
	• Vertical transmission of HIV ranges from 13% to 39% • Primary assays showed cytotoxic activity against at least one HIV protein was detected in 70% of infected children • Epitopes recognized in £ve children were mapped using synthetic peptides and secondary cultures • Patient EM28 (CDC P2A) had a CTL response to four epitopes in Gag				
p24(131–140)	p24(263–272)	KRWIILLGLNK	HIV-1 infection	human(B*27)	[Huang (2000)]
	• The single cell ELISPOT assay was optimized and highly speci£c, and found to work well even after the primary cells had been frozen and thawed • Increases in gamma interferon producing cells were observed in response to anti-retroviral therapy using single cell IFN-gamma-production ELISPOT • In 3/3 HLA A*02, B*27 individuals, the dominant response in gag measured by both gamma IFN production and T cell lysis was to the B27 epitope, KRWIILLGLNK, not the A2 SLYNTVATL epitope				
p24(131–140)	p24(263–272 SF2)	KRWIILGLNK	HIV-1 infection	human(B*27)	[McAdam (1998)]
	• Epitope invariant across clades A, B, C, and D				
p24(131–140)	p24(260–269 HIV-2)	RRWIQLGLQK		human(B*2703)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*2703 epitope				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–140)	p24()	KRWIILGGLNK	HIV-1 infection	human(B*2705)	[Wilson (2000)]
	• Three individuals with highly focused HIV-speci£c CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-speci£c CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-speci£c T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • Tetramers with peptide variants KRWIILGGLNK and KRWIIMGGLNK were used – CTL from most B27 donors recognize both variants, although one of the three subjects recognized only KRWIILGGLNK • ELISPOT was used to test a panel of CTL epitopes that had been de£ned earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWIILGGLNK • The subject with A*0201 had a moderatly strong strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL			
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B*2705)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*2705 epitope				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B*2705,B27)	[Goulder (1997b), Goulder (1997a)]
	• HLA-B*2705 is associated with slow HIV disease progression • 11/11 HLA-B*2705 donors make a response to this epitope, usually an immunodominant response • This is a highly conserved epitope • The HLA-B*2705 binding motif includes R at position 2, and L in the C-term position • [Goulder (1997a)] is a review on CTL immune escape that discusses this epitope in the context of the dif£culty in detection of immune escape – KRWIILGLNK and an R2K change, KKWIILGLNK, show little difference in titration curves, yet the K2 variants fail to bind to targets for more than 1 hour, while the R2 form can sensitize lysis by CTL for over 24 hours – minigene transfection experiments con£rmed the importance of this for the CTL response				
p24(131–140)	p24(260–269 HIV-2)	RRWIQLGLQK		human(B27)	[Brander & Walker(1996)]
	• HIV-2, HLA-B*2703, S. Rowland-Jones, Pers. Comm.				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B27)	[Fan (1997)]
	• The capacity of dendritic cells to process and present antigen and stimulate anti-HIV-1 CTL memory responses was studied				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–140)	Gag(263–272)	KRWIILGLNK	HIV-1 infection	human(B27)	[Zheng (1999)]
	• Protein delivery (gp160 LAV, p66 LAV, and p24 NY5) to human dendritic cells (DC) with liposomes provides enhanced memory CTL response relative to delivery of protein alone • Chloroquine administration enhanced epitope presentation, and brefeldin A and peptide aldehyde inhibitors inhibited antigen presentation, suggesting epitopes were processed by classical proteasome pathway • The CTL response to p24 was measured in individuals with a response to B27-KRWIILGLNK				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B27)	[Wilson (1998)]
	• HIV+ individuals were followed longitudinally using MHC tetramers in combination with 14 anti-BV chain MAbs, and clonal expansion of HIV-speci£c T cells was followed *in vivo* • Seven HIV+ people were studied, and all showed expansions of particular TCR BV clones, often several, relative to uninfected controls • Three patients were followed in detail, TCR VB expansions persisted for 2 to 3 years, with occasional transient increases				
p24(131–140)	p24()	KRWIILGLNK	HIV infection	human(B27)	[Rowland-Jones (1997)]
	• Described in this review as the £rst identi£ed HIV CTL epitope				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B27)	[Buseyne (1993b)]
	• Clustering of Gag p24 CTL epitopes recognized in 29 HIV-infected people				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B27)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes				
p24(131–140)	p24(263–272)	KRWIIMGLNK	HIV-1 infection	human(B27)	[Klenerman (1994)]
	• Naturally occurring variant KRWIILGLNK may act as antagonist				
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	• Naturally occurring variant KRWIILGLNK may act as antagonist				
p24(131–140)	p24(265–274)	KRWIILGLNK	HIV infection	human(B27)	[Moss (1995)]
	• In one individual, TCR usage changed over time indicating that new populations of CTL can be recruited • TCR usage showed a CTL clonal response to this epitope that persisted over 5 years • CTL clones speci£c for HIV epitopes may represent between 0.2 and 1% of the total CD8+ population of T cells				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–140)	p24(265–276)	KRWIILGLNK		human(B27)	[Carreno (1992)]
	• Included in HLA-B27 binding peptide competition study				
p24(131–140)	p24(265–274 SF2)	KRWIILGLNK	HIV-1 infection	human(B27)	[Phillips (1991), Goulder (1997a)]
	• Longitudinal study of CTL escape mutants – little variation was observed in the immunodominant B27 epitope, relative to B8 epitope • [Goulder (1997a)] is a review of immune escape that points out that there may be a protective effect associated with B27, and that HLA-B8 individuals tend to progress more rapidly than HLA B27 patients				
p24(131–140)	p24(263–272)	KRWIILGLNK	HIV-1 infection	human(B27)	[Nietfeld (1995), Goulder (1997a)]
	• Single point mutations were introduced and viral viability and CTL recognition tested – an Arg to Lys change at anchor position P2 abrogates binding to B27, but doesn„t change viral viability *in vitro* • [Goulder (1997a)] is a review of immune escape that summarizes this study				
p24(131–140)	p24(263–272)	KRWIIMGNK	HIV-1 infection	human(B27)	[Nowak (1995)]
	• Longitudinal study of CTL response and immune escape – the form KRWIILGNK was also found, and both forms stimulate CTL				
p24(131–140)	p24(263–272)	KRWIIMGLNK	HIV-1 infection	human(B27)	[Goulder (1997c), Goulder (1997a)]
	• Six HLA-B27 donors studied make a strong response to this epitope • In 4/6 cases, this was the immunodominant or only CTL response • Two of the cases had an epitope switch to the form KKWIIMGLNK during a period of rapid decline to AIDS, following their asymptomatic period • The arginine to lysine switch is in an anchor residue, and results in immune escape due to severely diminished binding to the B27 molecule • [Goulder (1997a)] is a review of immune escape that summarizes this study in the context of CTL escape to £xation				
p24(131–140)	p24()	KRWIILGLNK		human(B27)	[Rowland-Jones (1999)]
	• CTL responses in seronegative highly HIV-exposed African female sex workers in Gambia and Nairobi were studied – these women had no delta 32 deletion in CCR5 • In Gambia there is exposure to both HIV-1 and HIV-2, CTL responses to B35 epitopes in exposed, uninfected women are cross-reactive, and the B35 allele seems to be protective • HIV-2 sequence: RRWIQLGLQK – this epitope was not HIV-1 and HIV-2 cross-reactive				

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p24(131–140)	Gag()	KRWILGLNK	none, computer prediction	(B27)	[Schafer (1998)]
	• This study uses EpiMatrix for T cell epitope prediction to identify possible HLA-B27 and A-2 CTL epitopes in HIV • Based on EpiMatrix predictions, 28 peptides were synthesized and tested using T2 binding assays for potential HLA A2 or B27 binding, and 12 of these were shown to bind to the predicted HLA molecule • Two of these 12 peptides had been previously identi£ed as CTL epitopes: HLA-B27 KRWILGLNK and HLA-A2 ILKEPVHGV • This peptide sequence is not conserved between clades, but is found in most B clade isolates				
p24(131–140)	p24(263–282)	KRWIILGLNK	HIV-1 infection	human(B27)	[Bernard (1998)]
	• This study focuses on six rare long-term survivor HIV-infected people who were infected for many years without exhibiting immune dysregulation – such immunologically normal HIV-infected (INHI) cases occur at a frequency between 0.1 and 1% in the infected population • No direct CTL were found in any of the six INHIs, but above background CTLp activity was founded in 3/6 INHIs • Epitope sequences were deduced from larger reactive peptides based on HLA binding motifs – XRXXXXXXXK is a B*2705 binding motif				
p24(131–142)	p24(265–276)	KRWIILGLNKIV	no CTL shown	human(B27)	[Jardetzky (1991)]
	• Epitope examined in the context of peptide binding to HLA-B27				
p24(131–142)	p24(263–274 LAI)	KRWIILGLNKIV	HIV-1 infection	human(B27)	[Fan (1997)]
	• The capacity of dendritic cells to process and present antigen and stimulate anti-HIV-1 CTL memory responses was studied				
p24(131–145)	p24()	KRWILGLNKIVRMY	HIV-1 infection	human()	[Goulder (2000)]
	• The CTL-dominant response was focused on this epitope in a HIV+ African American living in Boston with unknown HLA – this epitope did not fall within the three most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p24(131–145)	p24(263–277 LAI)	KRWIILGLNKIVMRY	HIV-1 infection	human(A33)	[Buseyne (1993b)]
	• Clustering of Gag p24 CTL epitopes recognized in 29 HIV-infected people				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–145)	p24(266–277)	KRWIILGLNKIVRMY	rec gag-vaccinia	human(B27)	[Nixon (1988)]
	• Gag CTL epitope mapped with rec gag-vaccinia and synthetic peptides • This was the £rst HIV-1 epitope to be mapped				
p24(131–145)	p24(266–277 LAI)	KRWIILGLNKIVMRY	HIV-1 infection	human(B27)	[Meyerhans (1991)]
	• Longitudinal study showing persistence of epitope despite CTL activity				
p24(131–145)	p24(265–279)	KRWIILGLNKIVRMY	HIV-1 infection	human(B27)	[Nixon (1990), Rowland-Jones (1999)]
	• HIV-1 and HIV-2 cross-reactive CTL clone, highly conserved epitope • Reviewed in Rowland-Jones99, notes that it did not appear cross-reactive with HIV-2 in Rowland-Jones98, HIV-2 form: RRWIQL-GLQK				
p24(131–146)	p24(265–279)	KRWIILGLNKIVRMYC	HIV-1 infection	human(B27)	[Bouillot (1989)]
	• HLA-B27 restricted epitope also binds to HLA-A2 and HLA-B37 in solid phase assay				
p24(132–145)	Gag()	KWILGLNKIVRMY	HIV-infection	human()	[Weekes (1999a)]
	• Peptide 728: Memory CTL speci£c for HIV-1 may contribute to oligoclonal expansions within the CD57+ CD28- CD8+ CTLp populations				
p24(132–145)	Gag()	KWILGLNKIVRMY	HIV-infection	human(B27)	[Weekes (1999b)]
	• Peptide 728: Almost all CD8+ T cells are CD28+ at birth, and the proportion of CD28-CD8+ cells increases with age – this study examines the contribution of CD8+CD28- cells to CTL memory pools for CTL clones speci£c for two persistent human viruses, CMV and HIV – clones were found to be similarly distributed the CD28 depleted cell population • HIV CTL responses to 3 Env and 2 Gag peptides were studied • The clonal composition of the TCR Vbeta responses were studied and was found to be highly focused, with one TCR beta-chain sequence tending to dominate the peptide-speci£c response – clones to this epitope were Vbeta22.1				
p24(134–143)	p24()	IILGLNKIVR	HIV-1 exposure	human(A33)	[Rowland-Jones (1998b)]
	• HIV-speci£c CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes • This epitope is conserved among A, B and D clade viruses				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(136–145)	p24(268–277 LAI)	LGLNKIVRMY	Predicted from larger peptide	human(Bw62)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes • Also P. Johnson, Pers. Comm.				
p24(136–146)	p24(271–281)	LGLNKIVRMYS	HIV-1 infection	human(B62)	[Lubaki (1997)]
	• Eighty two HIV-1-speci£c CTL clones from 5 long-term non-progressors were isolated and analyzed for breadth of response • A sustained Gag, Env and Nef response was observed, and clones were restricted by multiple HLA epitopes, indicating a polyclonal response • A subject who was B62+ had CTL that recognized this peptide, p17 KIRLRPGGKKKYKL, and one additional unknown epitope • The two clones that recognized this epitope used two different Vβ genes, further demonstrating a polyclonal response				
p24(137–145)	p24()	GLNKIVRMY	HIV-1 infection	human()	[Goulder (2000)]
	• The CTL-dominant response was focused on this epitope in a HIV+ South African living in Durban, HLA A2/- B5802/62 Cw4/6 – this epitope did not fall within the three most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p24(137–145)	p24(272–280 LAI)	GLNKIVRMY	HIV-1 infection	human(B*1501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*1501 epitope				
p24(137–145)	p24(272–280 LAI)	GLNKIVRMY	HIV-1 infection	human(B62)	[Goulder (1997a)]
	• This paper is a review of CTL and immune evasion, but it presents a study of a shift from an HLA-A*0201 response to SLYNTVATL, to a B62 response to GLNKIVRMY • As long as a strong CTL response to SLYNTVATL was evident, the epitope variants SLFNTVATL or SLYNTIATL dominated the viral population – eventually the CTL response to the index peptide became undetectable, the CTL response shifted to a focus on GLNKIVRMY, and the index peptide SLYNTVATL once again established itself as the dominant form				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(137–145)	p24()	GLNKIVRMY	HIV-1 infection	human(B62)	[Goulder (2000)]

- The CTL-dominant response was focused on this epitope in a HIV+ African American living in Boston – this epitope did not fall within the three most recognized peptides in the study
- Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses
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Table 7: **All De£ned Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(129–136)	p24(263–270 SF2)	IYKRWIIL	HIV-1 infection	human(A*2402)	[Ikeda-Moore (1997)]
	• De£ned using reverse immunogenetics – 59 HLA-A*2402 binding peptides were predicted by searching for A*2402 anchors in HIV proteins (Tyr at 2, and Phe, Leu or Ile at the C term) – 53 of the 59 peptides bound A*2402 • This peptide induced CTL in 1/4 HIV-1+ people tested • IYKRWIIL bound to A*2402 with medium strength, the epitope can be processed in a vaccinia construct and presented – two speci£c CTL clones were obtained				
p24(129–138)	p24(263–272 SF2)	IYKRWIILGL	HIV-1 infection	human(A*2402)	[Ikeda-Moore (1997)]
	• De£ned using reverse immunogenetics – 59 HLA-A*2402 binding peptides were predicted by searching for A*2402 anchors in HIV proteins (Tyr at 2, and Phe, Leu or Ile at the C term) – 53 of the 59 peptides bound A*2402 • This peptide induced CTL in 1/4 HIV-1+ people tested • IYKRWIILGL bound to A*2402 with medium strength, the epitope can be processed in a vaccinia construct and presented – two speci£c CTL clones were obtained				
p24(129–138)	p24(263–272)	IYKRWIILGL	HIV-1 infection	human(B27)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned peptides from this database were used to screen for gamma interferon responses to other epitopes • 1/11 of the A2+ individuals was B27 and responded to IYKRWIILGL				
p24(130–148)	p24(265–280 BRU)	YKRWIILGLNKIVRMYSPT	HIV-1 infection	human(B27)	[Dadaglio (1991)]
	• Used as a positive control for HLA speci£city				
p24(131–139)	p24(263–272)	KRWIILGNK	HIV-1 infection	human(B27)	[Durali (1998)]
	• Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • One of the patients was shown to react to this epitope: KRWIILGNK				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–139)	Gag(265–273)	KRWIILGLN	HIV-1 infection	chimpanzee(Patr-B*03)	[Balla-Jhagjhoorsingh (1999)]
	• Certain HLA-alleles have been associated with long-term survival – among them are HLA-B*27 and HLA-B*57 • Of more than 150 chimpanzees that have been reported to be infected with HIV-1, only one has developed AIDS • CTL responses were studied in two HIV-1 infected chimpanzees that have strong CTL responses, and they were found to respond to highly conserved epitopes that are recognized in humans in the context of HLA-B*27 and HLA-B*57 • The human HLA protein which presents this Patr-B*03 epitope is HLA B*2705 but the amino acid sequences in the binding pockets of HLA-B*2705 and Patr-B*03 are distinctive				
p24(131–140)	Gag(263–272 LAI)	KRWILLGLNK	HIV-1 infection	human()	[Buseyne (1993a)]
	• Vertical transmission of HIV ranges from 13% to 39% • Primary assays showed cytotoxic activity against at least one HIV protein was detected in 70% of infected children • Epitopes recognized in £ve children were mapped using synthetic peptides and secondary cultures • Patient EM28 (CDC P2A) had a CTL response to four epitopes in Gag				
p24(131–140)	p24(263–272)	KRWIILLGLNK	HIV-1 infection	human(B*27)	[Huang (2000)]
	• The single cell ELISPOT assay was optimized and highly speci£c, and found to work well even after the primary cells had been frozen and thawed • Increases in gamma interferon producing cells were observed in response to anti-retroviral therapy using single cell IFN-gamma-production ELISPOT • In 3/3 HLA A*02, B*27 individuals, the dominant response in gag measured by both gamma IFN production and T cell lysis was to the B27 epitope, KRWIILLGLNK, not the A2 SLYNTVATL epitope				
p24(131–140)	p24(263–272 SF2)	KRWIILGLNK	HIV-1 infection	human(B*27)	[McAdam (1998)]
	• Epitope invariant across clades A, B, C, and D				
p24(131–140)	p24(260–269 HIV-2)	RRWIQLGLQK		human(B*2703)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*2703 epitope				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–140)	p24()	KRWIILGGLNK	HIV-1 infection	human(B*2705)	[Wilson (2000)]
	• Three individuals with highly focused HIV-speci£c CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-speci£c CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-speci£c T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • Tetramers with peptide variants KRWIILGGLNK and KRWIIMGGLNK were used – CTL from most B27 donors recognize both variants, although one of the three subjects recognized only KRWIILGGLNK • ELISPOT was used to test a panel of CTL epitopes that had been de£ned earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWIILGGLNK • The subject with A*0201 had a moderatly strong strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL			
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B*2705)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*2705 epitope				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B*2705,B27)	[Goulder (1997b), Goulder (1997a)]
	• HLA-B*2705 is associated with slow HIV disease progression • 11/11 HLA-B*2705 donors make a response to this epitope, usually an immunodominant response • This is a highly conserved epitope • The HLA-B*2705 binding motif includes R at position 2, and L in the C-term position • [Goulder (1997a)] is a review on CTL immune escape that discusses this epitope in the context of the dif£culty in detection of immune escape – KRWIILGLNK and an R2K change, KKWIILGLNK, show little difference in titration curves, yet the K2 variants fail to bind to targets for more than 1 hour, while the R2 form can sensitize lysis by CTL for over 24 hours – minigene transfection experiments con£rmed the importance of this for the CTL response				
p24(131–140)	p24(260–269 HIV-2)	RRWIQLGLQK		human(B27)	[Brander & Walker(1996)]
	• HIV-2, HLA-B*2703, S. Rowland-Jones, Pers. Comm.				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B27)	[Fan (1997)]
	• The capacity of dendritic cells to process and present antigen and stimulate anti-HIV-1 CTL memory responses was studied				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–140)	Gag(263–272)	KRWIILGLNK	HIV-1 infection	human(B27)	[Zheng (1999)]
	• Protein delivery (gp160 LAV, p66 LAV, and p24 NY5) to human dendritic cells (DC) with liposomes provides enhanced memory CTL response relative to delivery of protein alone • Chloroquine administration enhanced epitope presentation, and brefeldin A and peptide aldehyde inhibitors inhibited antigen presentation, suggesting epitopes were processed by classical proteasome pathway • The CTL response to p24 was measured in individuals with a response to B27-KRWIILGLNK				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B27)	[Wilson (1998)]
	• HIV+ individuals were followed longitudinally using MHC tetramers in combination with 14 anti-BV chain MAbs, and clonal expansion of HIV-speci£c T cells was followed *in vivo* • Seven HIV+ people were studied, and all showed expansions of particular TCR BV clones, often several, relative to uninfected controls • Three patients were followed in detail, TCR VB expansions persisted for 2 to 3 years, with occasional transient increases				
p24(131–140)	p24()	KRWIILGLNK	HIV infection	human(B27)	[Rowland-Jones (1997)]
	• Described in this review as the £rst identi£ed HIV CTL epitope				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B27)	[Buseyne (1993b)]
	• Clustering of Gag p24 CTL epitopes recognized in 29 HIV-infected people				
p24(131–140)	p24(263–272 LAI)	KRWIILGLNK	HIV-1 infection	human(B27)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes				
p24(131–140)	p24(263–272)	KRWIIMGLNK	HIV-1 infection	human(B27)	[Klenerman (1994)]
	• Naturally occurring variant KRWIILGLNK may act as antagonist				
p24(131–140)	p24(263–272)	KRWIIMGLNK	HIV-1 infection	human(B27)	[Klenerman (1995)]
	• Naturally occurring variant KRWIILGLNK may act as antagonist				
p24(131–140)	p24(265–274)	KRWIILGLNK	HIV infection	human(B27)	[Moss (1995)]
	• In one individual, TCR usage changed over time indicating that new populations of CTL can be recruited • TCR usage showed a CTL clonal response to this epitope that persisted over 5 years • CTL clones speci£c for HIV epitopes may represent between 0.2 and 1% of the total CD8+ population of T cells				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–140)	p24(265–276)	KRWIILGLNK		human(B27)	[Carreno (1992)]
	• Included in HLA-B27 binding peptide competition study				
p24(131–140)	p24(265–274 SF2)	KRWIILGLNK	HIV-1 infection	human(B27)	[Phillips (1991), Goulder (1997a)]
	• Longitudinal study of CTL escape mutants – little variation was observed in the immunodominant B27 epitope, relative to B8 epitope • [Goulder (1997a)] is a review of immune escape that points out that there may be a protective effect associated with B27, and that HLA-B8 individuals tend to progress more rapidly than HLA B27 patients				
p24(131–140)	p24(263–272)	KRWIILGLNK	HIV-1 infection	human(B27)	[Nietfeld (1995), Goulder (1997a)]
	• Single point mutations were introduced and viral viability and CTL recognition tested – an Arg to Lys change at anchor position P2 abrogates binding to B27, but doesn„t change viral viability *in vitro* • [Goulder (1997a)] is a review of immune escape that summarizes this study				
p24(131–140)	p24(263–272)	KRWIIMGNK	HIV-1 infection	human(B27)	[Nowak (1995)]
	• Longitudinal study of CTL response and immune escape – the form KRWIILGNK was also found, and both forms stimulate CTL				
p24(131–140)	p24(263–272)	KRWIIMGLNK	HIV-1 infection	human(B27)	[Goulder (1997c), Goulder (1997a)]
	• Six HLA-B27 donors studied make a strong response to this epitope • In 4/6 cases, this was the immunodominant or only CTL response • Two of the cases had an epitope switch to the form KKWIIMGLNK during a period of rapid decline to AIDS, following their asymptomatic period • The arginine to lysine switch is in an anchor residue, and results in immune escape due to severely diminished binding to the B27 molecule • [Goulder (1997a)] is a review of immune escape that summarizes this study in the context of CTL escape to £xation				
p24(131–140)	p24()	KRWIILGLNK		human(B27)	[Rowland-Jones (1999)]
	• CTL responses in seronegative highly HIV-exposed African female sex workers in Gambia and Nairobi were studied – these women had no delta 32 deletion in CCR5 • In Gambia there is exposure to both HIV-1 and HIV-2, CTL responses to B35 epitopes in exposed, uninfected women are cross-reactive, and the B35 allele seems to be protective • HIV-2 sequence: RRWIQLGLQK – this epitope was not HIV-1 and HIV-2 cross-reactive				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–140)	Gag()	KRWILGLNK	none, computer prediction	(B27)	[Schafer (1998)]
	• This study uses EpiMatrix for T cell epitope prediction to identify possible HLA-B27 and A-2 CTL epitopes in HIV • Based on EpiMatrix predictions, 28 peptides were synthesized and tested using T2 binding assays for potential HLA A2 or B27 binding, and 12 of these were shown to bind to the predicted HLA molecule • Two of these 12 peptides had been previously identi£ed as CTL epitopes: HLA-B27 KRWILGLNK and HLA-A2 ILKEPVHGV • This peptide sequence is not conserved between clades, but is found in most B clade isolates				
p24(131–140)	p24(263–282)	KRWIILGLNK	HIV-1 infection	human(B27)	[Bernard (1998)]
	• This study focuses on six rare long-term survivor HIV-infected people who were infected for many years without exhibiting immune dysregulation – such immunologically normal HIV-infected (INHI) cases occur at a frequency between 0.1 and 1% in the infected population • No direct CTL were found in any of the six INHIs, but above background CTLp activity was founded in 3/6 INHIs • Epitope sequences were deduced from larger reactive peptides based on HLA binding motifs – XRXXXXXXXK is a B*2705 binding motif				
p24(131–142)	p24(265–276)	KRWIILGLNKIV	no CTL shown	human(B27)	[Jardetzky (1991)]
	• Epitope examined in the context of peptide binding to HLA-B27				
p24(131–142)	p24(263–274 LAI)	KRWIILGLNKIV	HIV-1 infection	human(B27)	[Fan (1997)]
	• The capacity of dendritic cells to process and present antigen and stimulate anti-HIV-1 CTL memory responses was studied				
p24(131–145)	p24()	KRWILGLNKIVRMY	HIV-1 infection	human()	[Goulder (2000)]
	• The CTL-dominant response was focused on this epitope in a HIV+ African American living in Boston with unknown HLA – this epitope did not fall within the three most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p24(131–145)	p24(263–277 LAI)	KRWIILGLNKIVMRY	HIV-1 infection	human(A33)	[Buseyne (1993b)]
	• Clustering of Gag p24 CTL epitopes recognized in 29 HIV-infected people				

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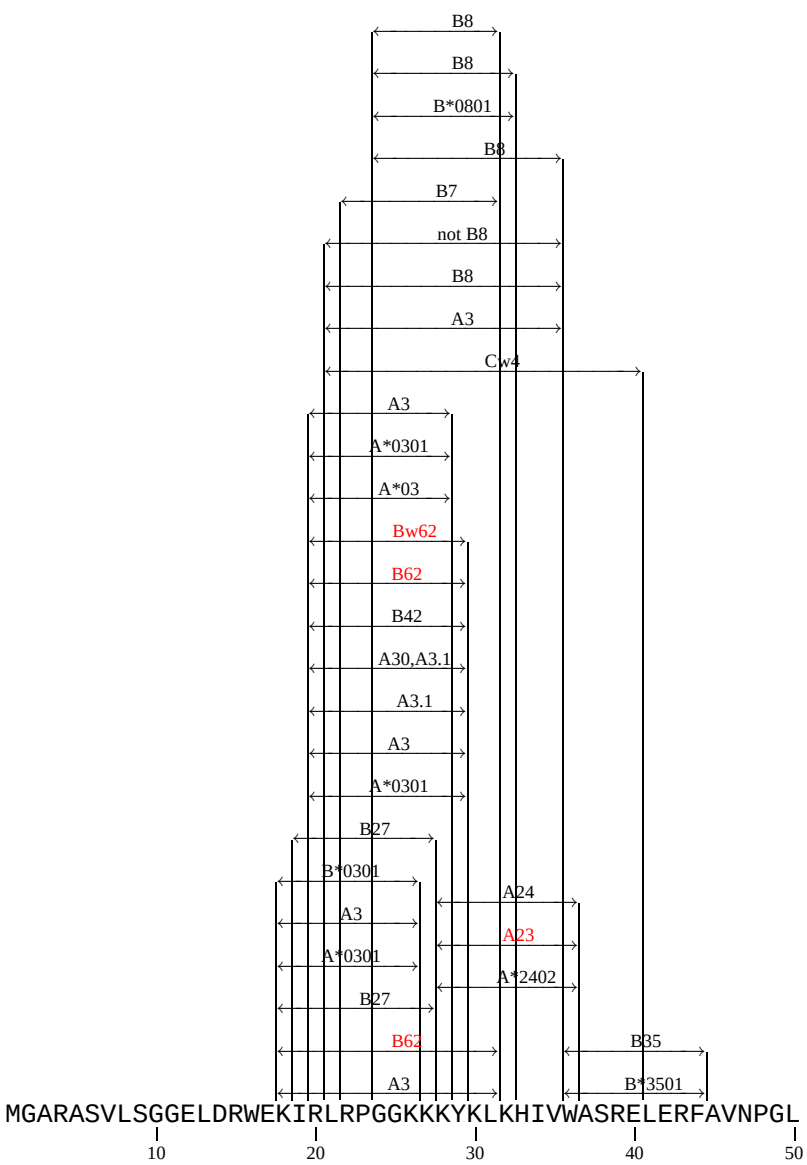
	• Gag CTL epitope mapped with rec gag-vaccinia and synthetic peptides • This was the £rst HIV-1 epitope to be mapped				
p24(131–145)	p24(266–277 LAI)	KRWIILGLNKIVMRY	HIV-1 infection	human(B27)	[Meyerhans (1991)]
	• Longitudinal study showing persistence of epitope despite CTL activity				
p24(131–145)	p24(265–279)	KRWIILGLNKIVRMY	HIV-1 infection	human(B27)	[Nixon (1990), Rowland-Jones (1999)]
	• HIV-1 and HIV-2 cross-reactive CTL clone, highly conserved epitope • Reviewed in Rowland-Jones99, notes that it did not appear cross-reactive with HIV-2 in Rowland-Jones98, HIV-2 form: RRWIQL-GLQK				
p24(131–146)	p24(265–279)	KRWIILGLNKIVRMYC	HIV-1 infection	human(B27)	[Bouillot (1989)]
	• HLA-B27 restricted epitope also binds to HLA-A2 and HLA-B37 in solid phase assay				
p24(132–145)	Gag()	KWILGLNKIVRMY	HIV-infection	human()	[Weekes (1999a)]
	• Peptide 728: Memory CTL speci£c for HIV-1 may contribute to oligoclonal expansions within the CD57+ CD28- CD8+ CTLp populations				
p24(132–145)	Gag()	KWILGLNKIVRMY	HIV-infection	human(B27)	[Weekes (1999b)]
	• Peptide 728: Almost all CD8+ T cells are CD28+ at birth, and the proportion of CD28-CD8+ cells increases with age – this study examines the contribution of CD8+CD28- cells to CTL memory pools for CTL clones speci£c for two persistent human viruses, CMV and HIV – clones were found to be similarly distributed the CD28 depleted cell population • HIV CTL responses to 3 Env and 2 Gag peptides were studied • The clonal composition of the TCR Vbeta responses were studied and was found to be highly focused, with one TCR beta-chain sequence tending to dominate the peptide-speci£c response – clones to this epitope were Vbeta22.1				
p24(134–143)	p24()	IILGLNKIVR	HIV-1 exposure	human(A33)	[Rowland-Jones (1998b)]
	• HIV-speci£c CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes • This epitope is conserved among A, B and D clade viruses				

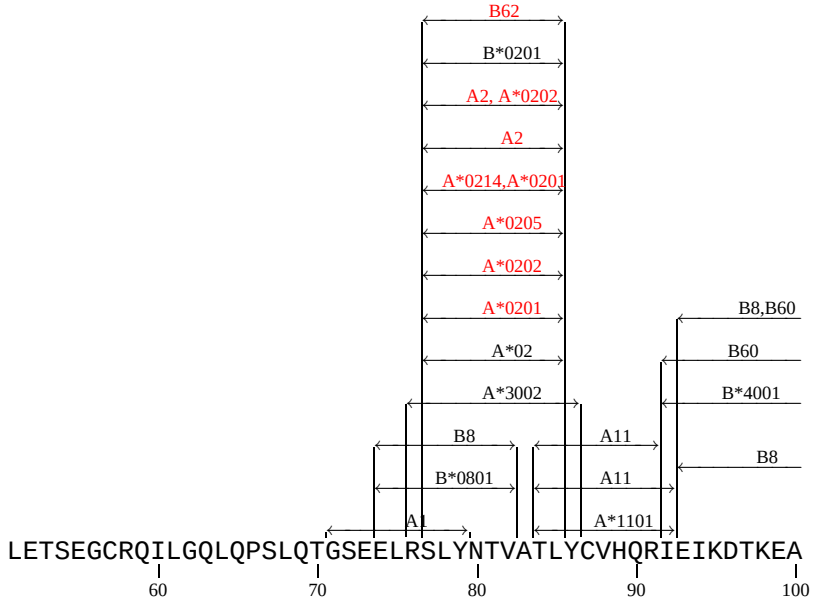
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(136–145)	p24(268–277 LAI)	LGLNKIVRMY	Predicted from larger peptide	human(Bw62)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes • Also P. Johnson, Pers. Comm.				
p24(136–146)	p24(271–281)	LGLNKIVRMYS	HIV-1 infection	human(B62)	[Lubaki (1997)]
	• Eighty two HIV-1-speci£c CTL clones from 5 long-term non-progressors were isolated and analyzed for breadth of response • A sustained Gag, Env and Nef response was observed, and clones were restricted by multiple HLA epitopes, indicating a polyclonal response • A subject who was B62+ had CTL that recognized this peptide, p17 KIRLRPGGKKKYKL, and one additional unknown epitope • The two clones that recognized this epitope used two different Vβ genes, further demonstrating a polyclonal response				
p24(137–145)	p24()	GLNKIVRMY	HIV-1 infection	human()	[Goulder (2000)]
	• The CTL-dominant response was focused on this epitope in a HIV+ South African living in Durban, HLA A2/- B5802/62 Cw4/6 – this epitope did not fall within the three most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p24(137–145)	p24(272–280 LAI)	GLNKIVRMY	HIV-1 infection	human(B*1501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*1501 epitope				
p24(137–145)	p24(272–280 LAI)	GLNKIVRMY	HIV-1 infection	human(B62)	[Goulder (1997a)]
	• This paper is a review of CTL and immune evasion, but it presents a study of a shift from an HLA-A*0201 response to SLYNTVATL, to a B62 response to GLNKIVRMY • As long as a strong CTL response to SLYNTVATL was evident, the epitope variants SLFNTVATL or SLYNTIATL dominated the viral population – eventually the CTL response to the index peptide became undetectable, the CTL response shifted to a focus on GLNKIVRMY, and the index peptide SLYNTVATL once again established itself as the dominant form				

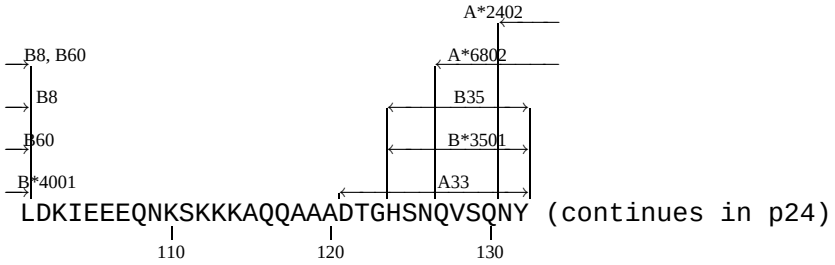
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(137–145)	p24()	GLNKIVRMY	HIV-1 infection	human(B62)	[Goulder (2000)]

- The CTL-dominant response was focused on this epitope in a HIV+ African American living in Boston – this epitope did not fall within the three most recognized peptides in the study
- Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses
- Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa

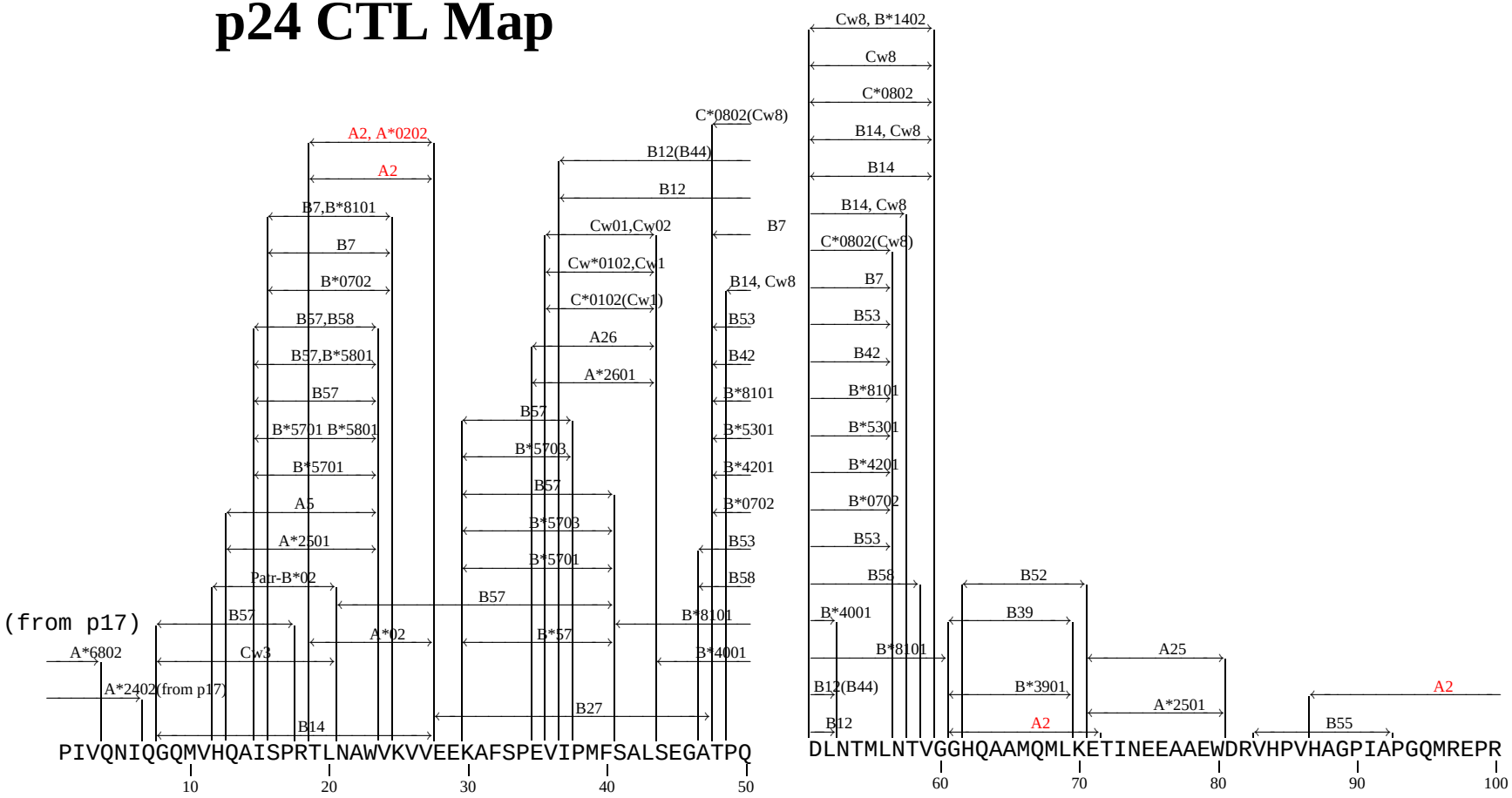
p17 CTL Map

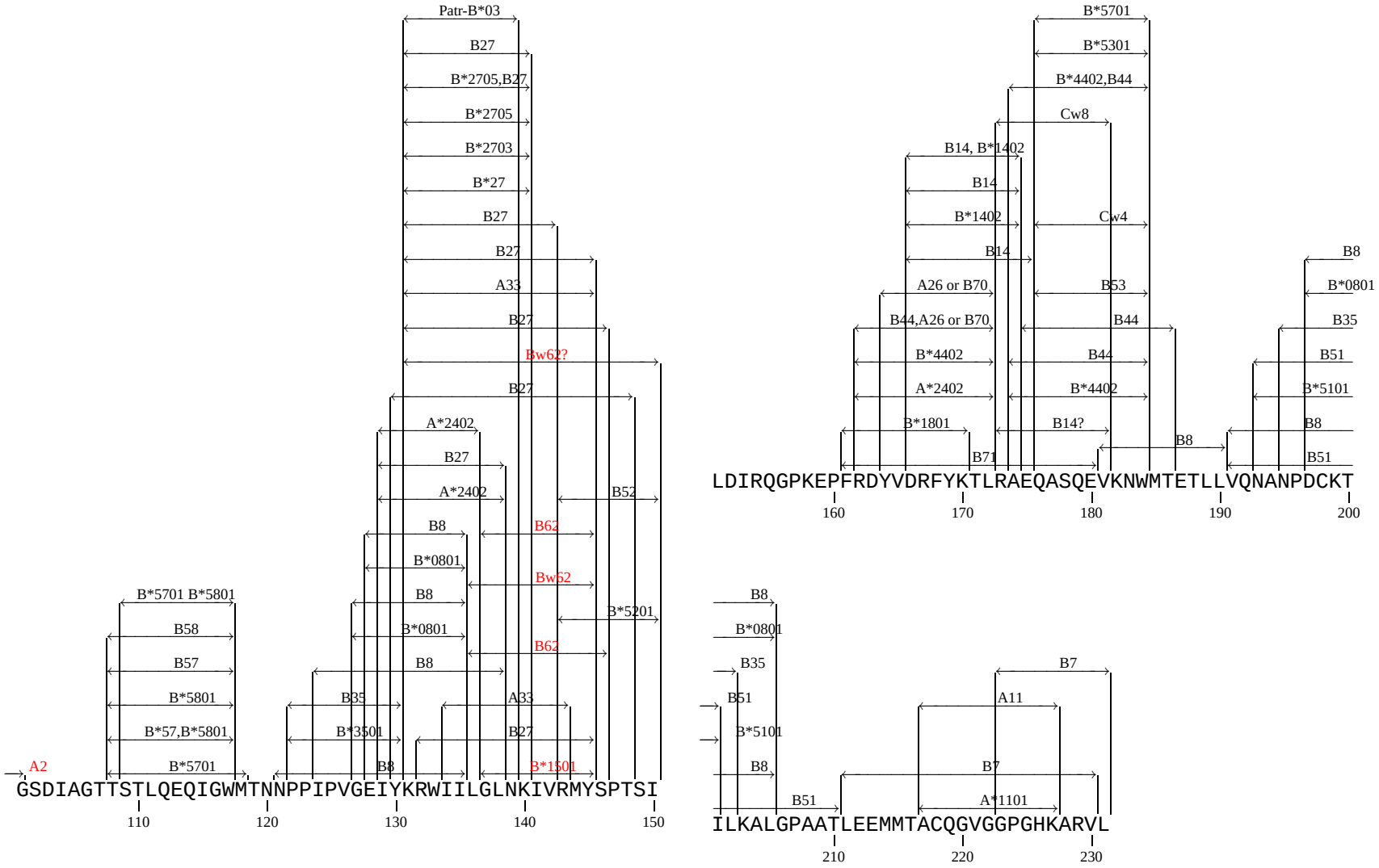

B8
B8
B*0801
B8
B7
not B8
B8
A3
Cw4
A3
A*0301
A*03
Bw62
B62
B42
A30,A3.1
A3.1
A3
A*0301
B27
B*0301
A3
A*0301
B27
B62
A3
A24
A23
A*2402
B35
B*3501
MGARASVLSGGELDRWEKIRLRPGGKKKYKLKHIVWASRELERFAVNPGL
10
20
30
40
50


B62
B*0201
A2, A*0202
A2
A*0214,A*0201
A*0205
A*0202
A*0201
A*02
A*3002
B8
B*0801
A1
A11
A11
A*1101
B8,B60
B60
B*4001
B8
LETSEGCRQILGQLQPSLQTGSEELRSLYNTVATLYCVHQRIEIKDTKEA
60
70
80
90
100

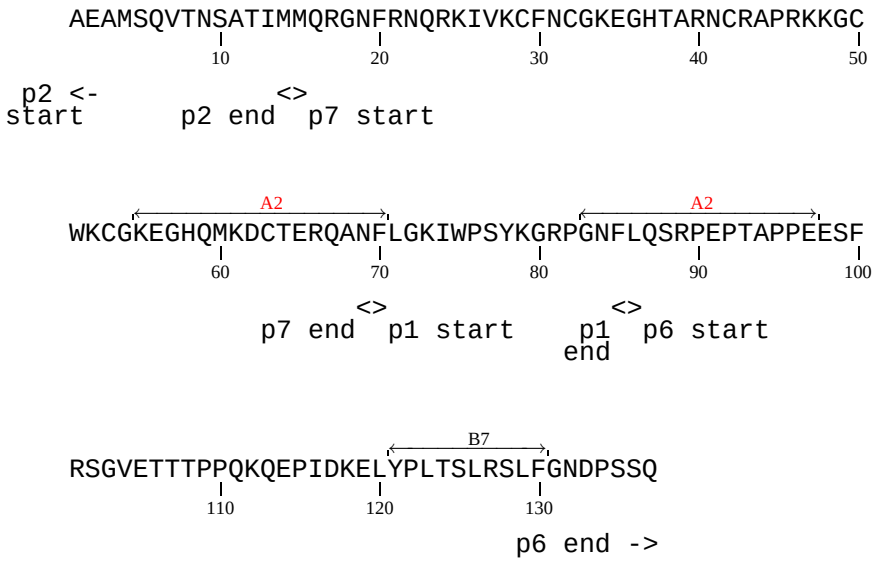

A*2402
A*6802
B35
B*3501
A33
B8, B60
B8
B60
B*4001
LDKIEEEQNKSKKKAQQAAADTGHSNQVSQNY (continues in p24)
110
120
130

p24 CTL Map

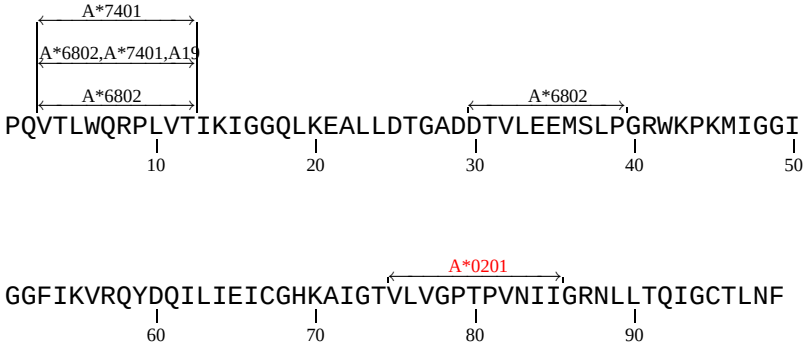

(from p17)
A*6802
A*2402(from p17)
A2, A*0202
A2
B7,B*8101
B7
B*0702
B57,B58
B57,B*5801
B57
B*5701 B*5801
B*5701
A5
A*2501
Pair-B*02
B57
A*02
Cw3
B14
C*0802(Cw8)
B12(B44)
B12
Cw01,Cw02
Cw*0102,Cw1
C*0102(Cw1)
A26
A*2601
B57
B*5703
B57
B*5703
B*5701
B57
B*57
B27
B7
B14, Cw8
B53
B42
B*8101
B*5301
B*4201
B*0702
B53
B58
B*8101
B*4001
Cw8, B*1402
Cw8
C*0802
B14, Cw8
B14
B14, Cw8
C*0802(Cw8)
B7
B53
B42
B*8101
B*5301
B*4201
B*0702
B53
B58
B*4001
B*8101
B12(B44)
B12
B52
B39
B*3901
A2
A25
A*2501
A2
B55
PIVQNIQGQMVHQAISPRTLNAWVKVVEEKAFSPEVIPMFSALSEGATPQ
DLNTMLNTVGGHQAAMQMLKETINEEAAEWDRVHPVHAGPIAPGQMREPR
10
20
30
40
50
60
70
80
90
100


Patr-B*03
B27
B*2705,B27
B*2705
B*2703
B*27
B27
B27
A33
B27
Bw62?
B27
A*2402
B27
A*2402
B52
B8
B62
B*0801
Bw62
B*5701 B*5801
B8
B*5201
B58
B*0801
B62
B57
B8
B*5801
B35
A33
B*57,B*5801
B*3501
B27
A2
B*5701
B8
B*1501
GSDIAGTTSTLQEQIGWMTNNPPIPVGEIYKRWIILGLNKIVRMYSPTSI
110
120
130
140
150
B*5701
B*5301
B*4402,B44
Cw8
B14, B*1402
B14
B*1402
Cw4
B14
B8
A26 or B70
B53
B*0801
B44,A26 or B70
B44
B35
B*4402
B44
B51
A*2402
B*4402
B*5101
B*1801
B14?
B8
B8
B71
B51
LDIRQGPKEPFRDYVDRFYKTLRAEQASQEVKNWMTETLLVQNANPDCKT
160
170
180
190
200
B8
B*0801
B35
B51
B*5101
B8
B51
B7
A11
B7
A*1101
ILKALGPAATLEEMMTACQGVGGPGHKARVL
210
220
230

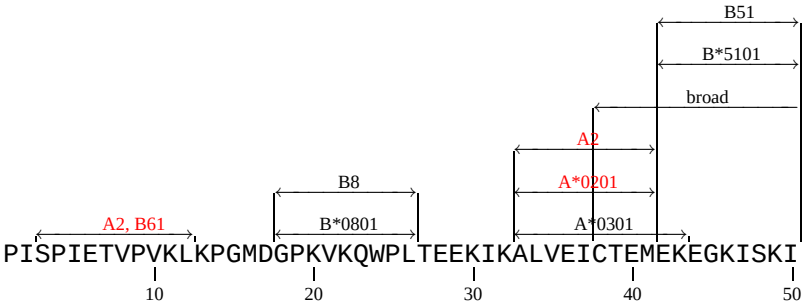
p2p7p1p6 CTL Map

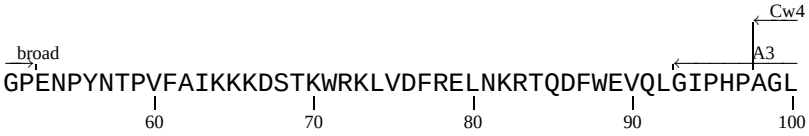


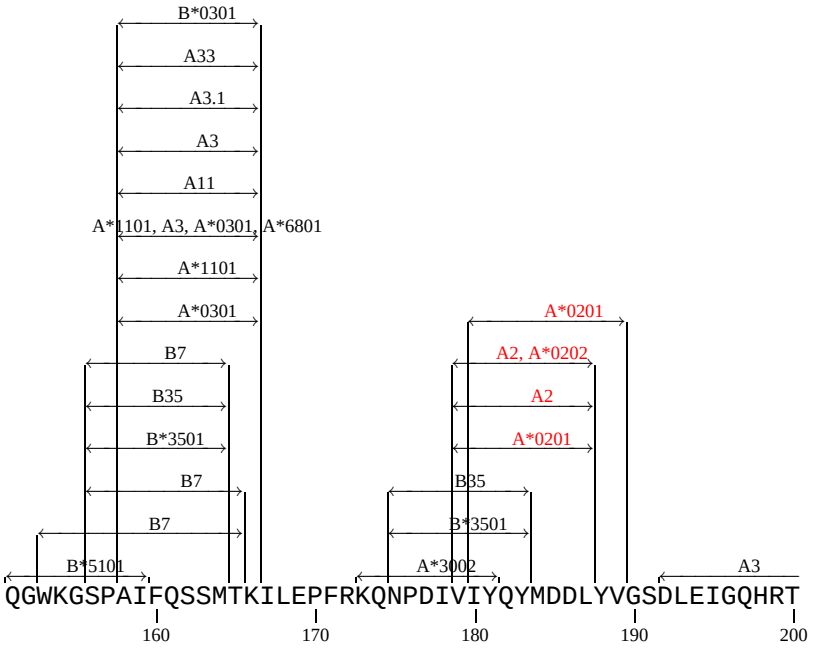
Protease CTL Map

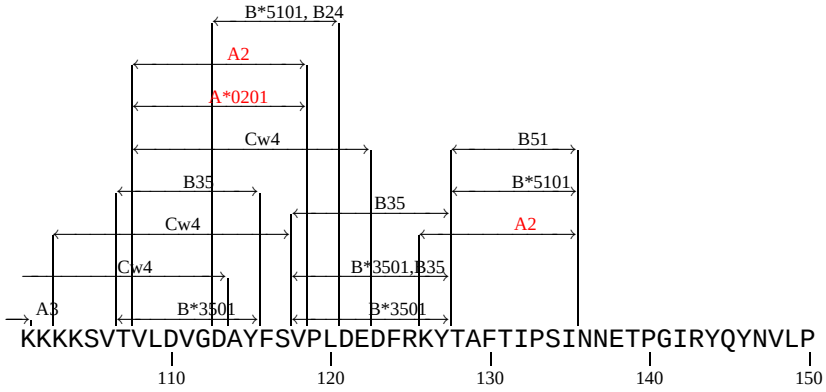


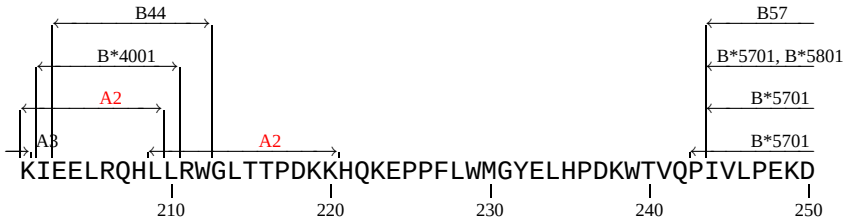
RT CTL Map

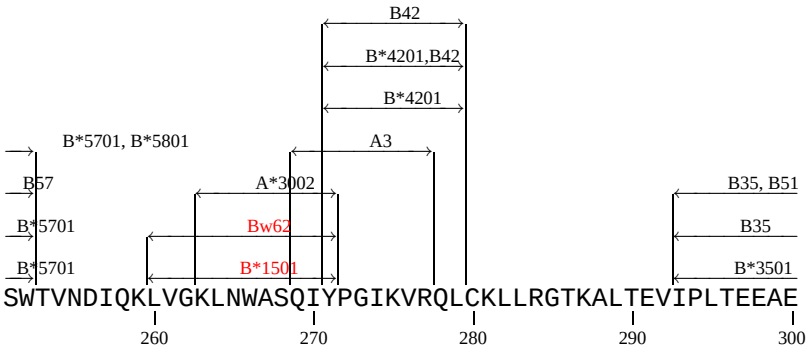

B51
B*5101
broad
A2
B8
A*0201
A2, B61
B*0801
A*0301
PISPIETVPVKLKPGMDGPKVKQWPLTEEKIKALVEICTEMEKEGKISKI
10
20
30
40
50

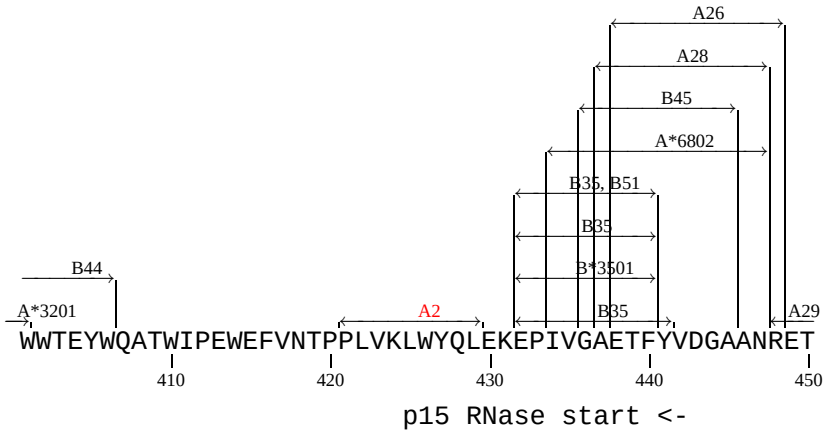

Cw4
broad
A3
GPENPYNTPVFAIKKKDSTKWRKLVDFRELNKRTQDFWEVQLGIPHPAGL
60
70
80
90
100


B*0301
A33
A3.1
A3
A11
A*1101, A3, A*0301, A*6801
A*1101
A*0301
B7
B35
B*3501
B7
B7
B*5101
A*0201
A2, A*0202
A2
A*0201
B35
B*3501
A*3002
A3
QGWKGSPAIFQSSMTKILEPFRKQNPDIVIYQYMDDLYVGSDLEIGQHRT
160
170
180
190
200

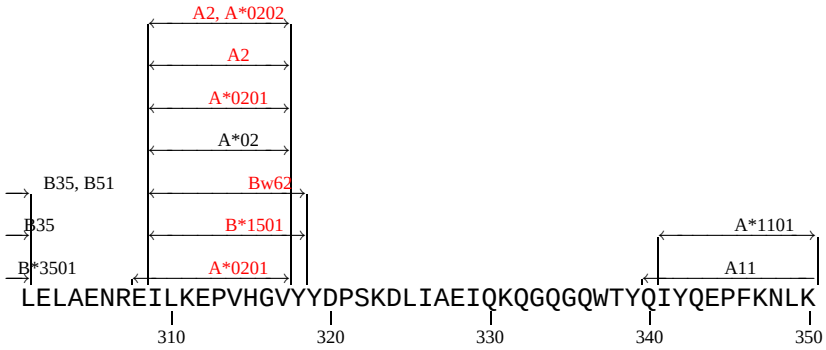

B*5101, B24
A2
A*0201
Cw4
B51
B35
B*5101
B35
Cw4
A2
Cw4
B*3501,B35
A3
B*3501
B*3501
KKKKSVTVLDVGDAYFSVPLDEDFRKYTAFTIPSINNETPGIRYQYNVLP
110
120
130
140
150

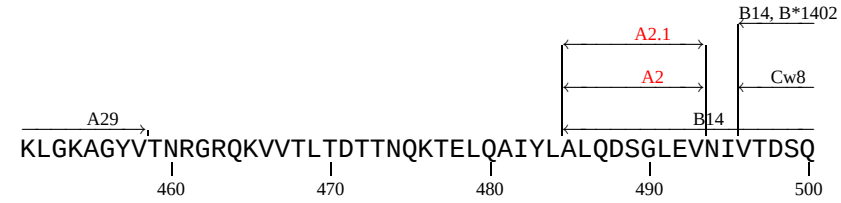

B44
B57
B*4001
B*5701, B*5801
A2
B*5701
A3
A2
B*5701
KIEELRQHLLRWGLTTPDKKHQKEPPFLWMGYELHPDKWTVQPIVLPEKD
210
220
230
240
250

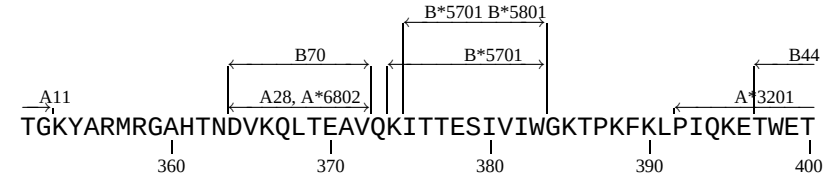


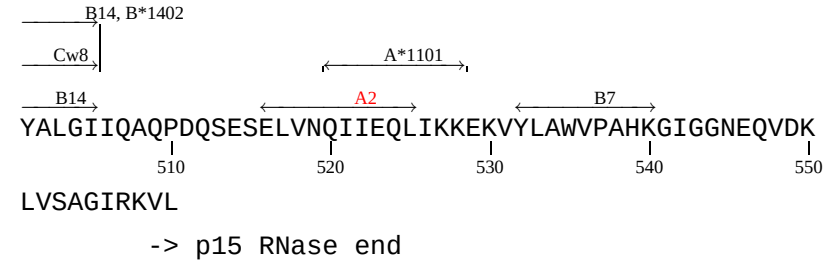


p15 RNase start <-





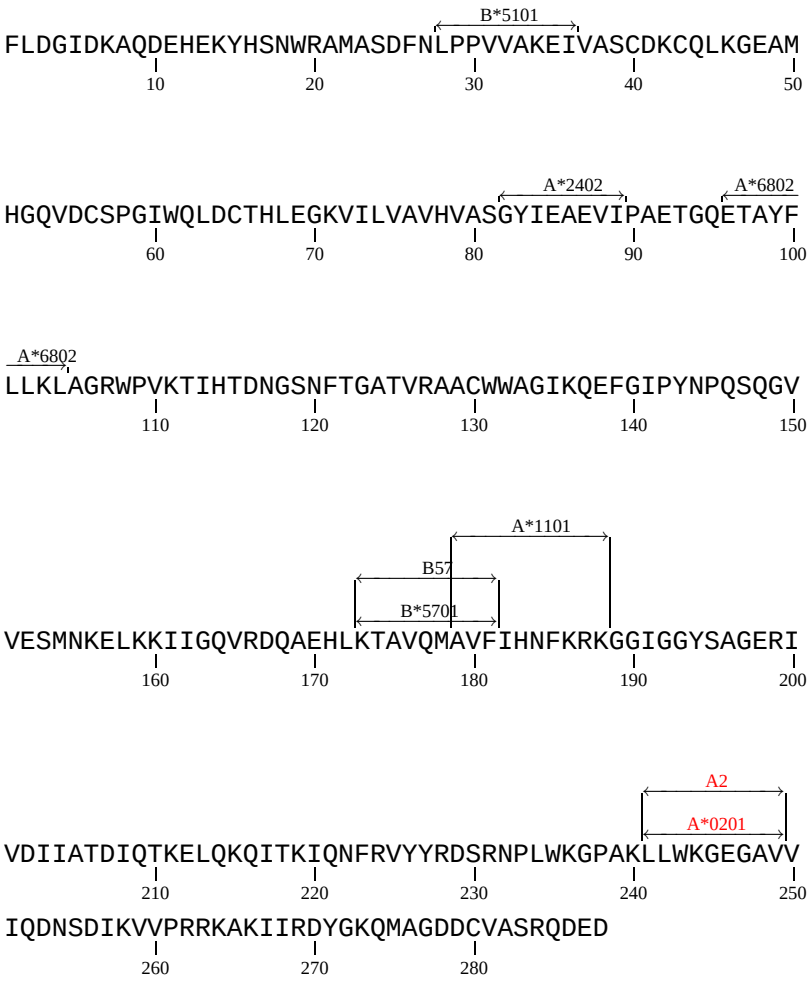




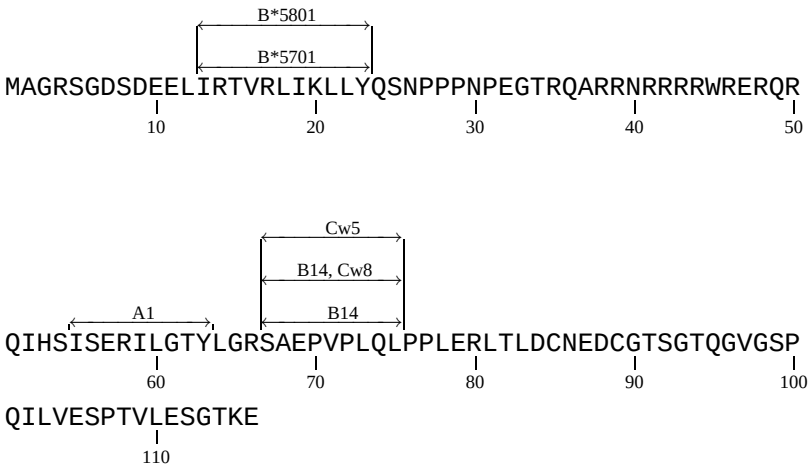
LVSAGIRKVL

-> p15 RNase end

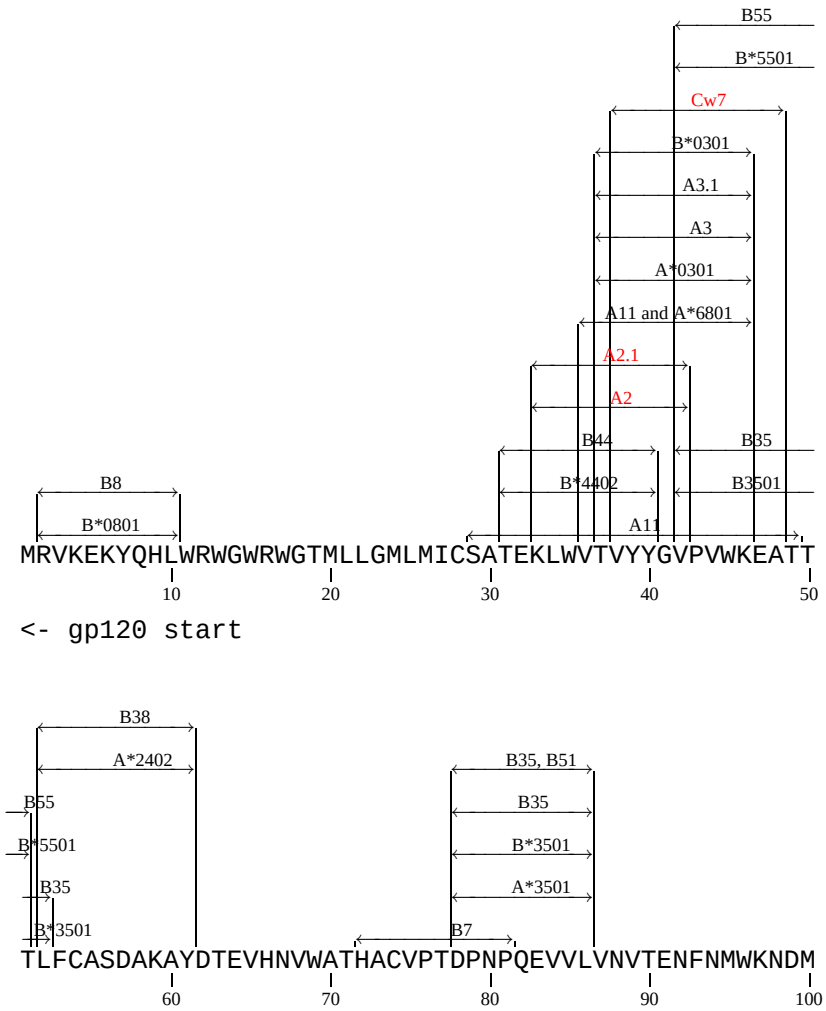
Integrase CTL Map



Rev CTL Map



gp160 CTL Map



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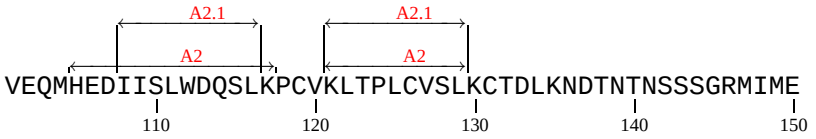
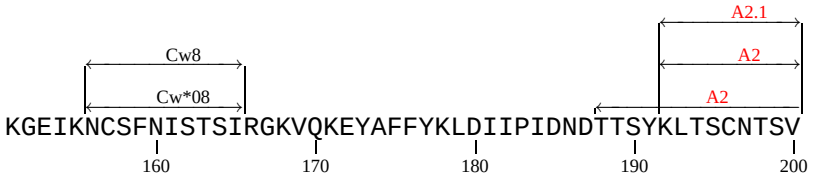
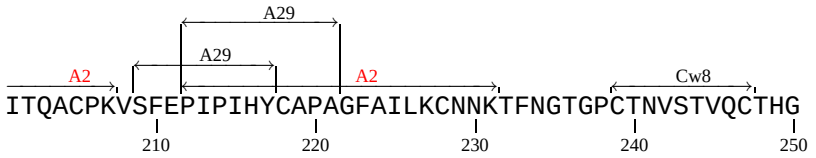
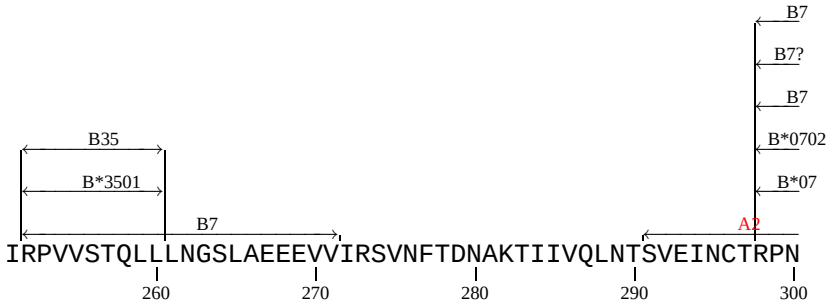
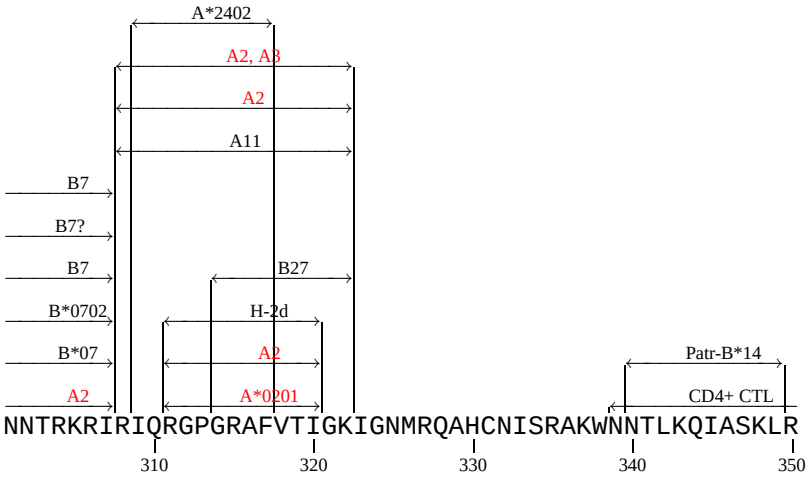
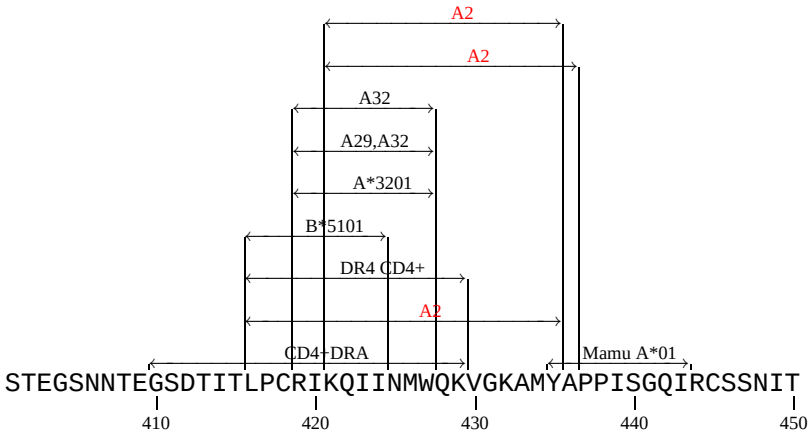




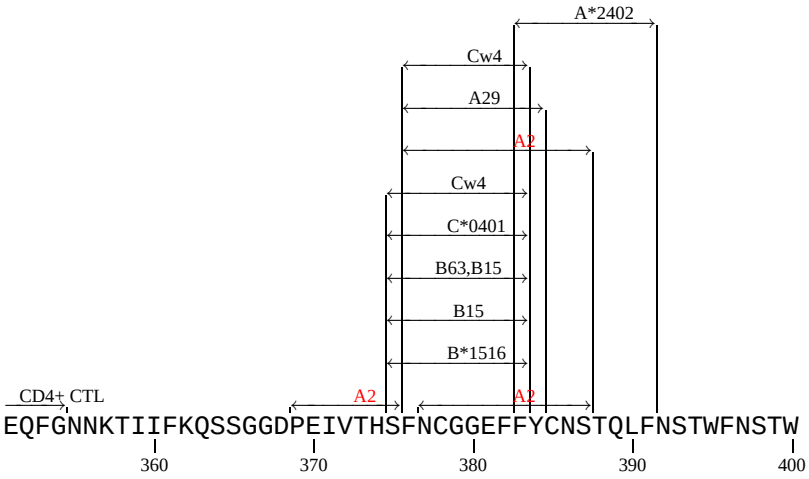


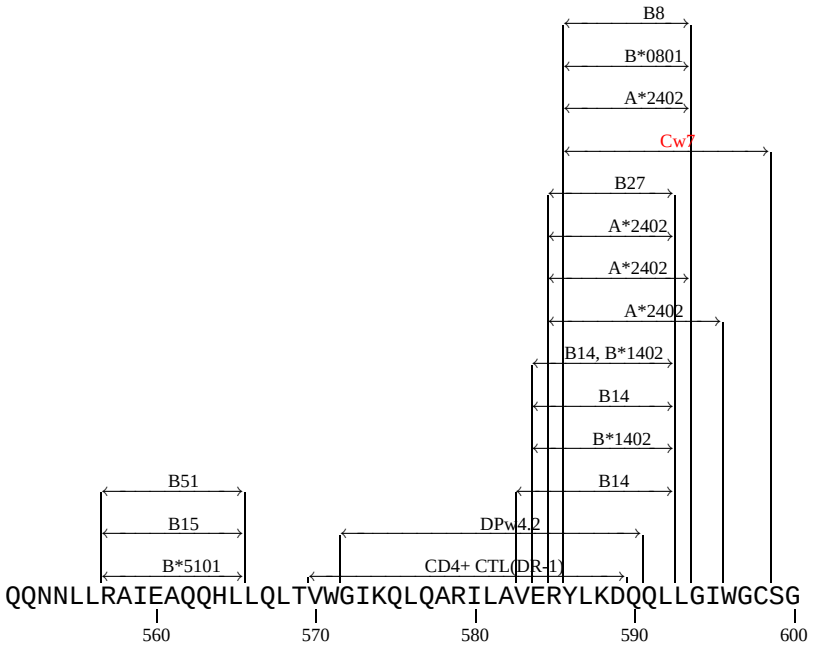


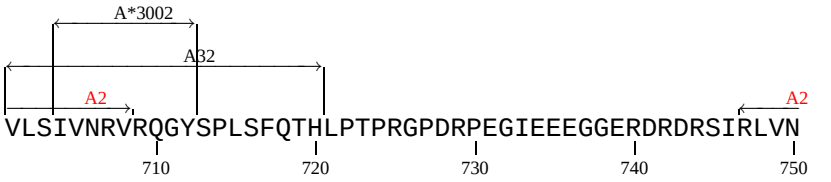


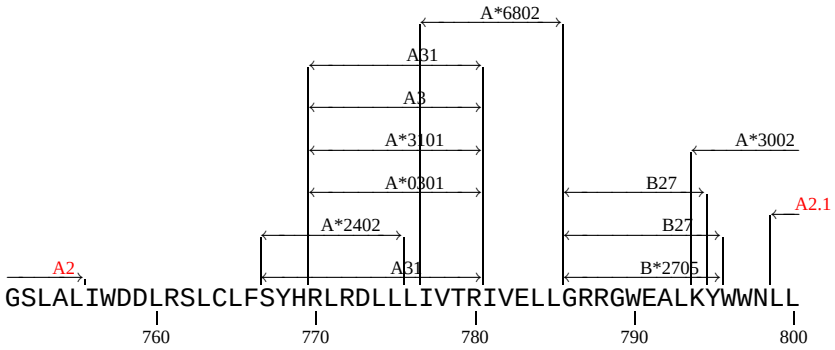


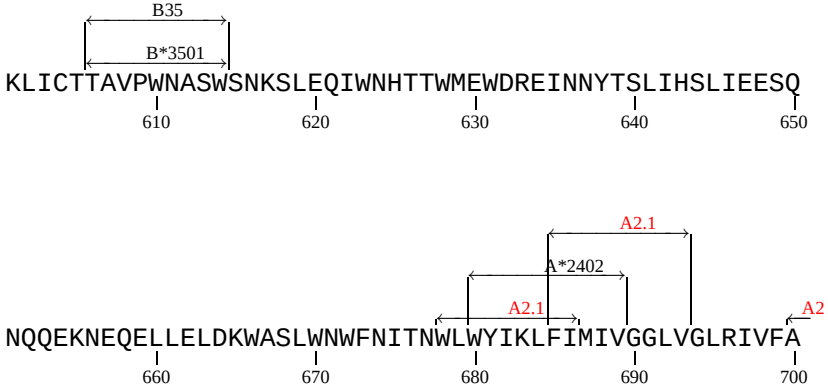
gp120 end <> gp41 start



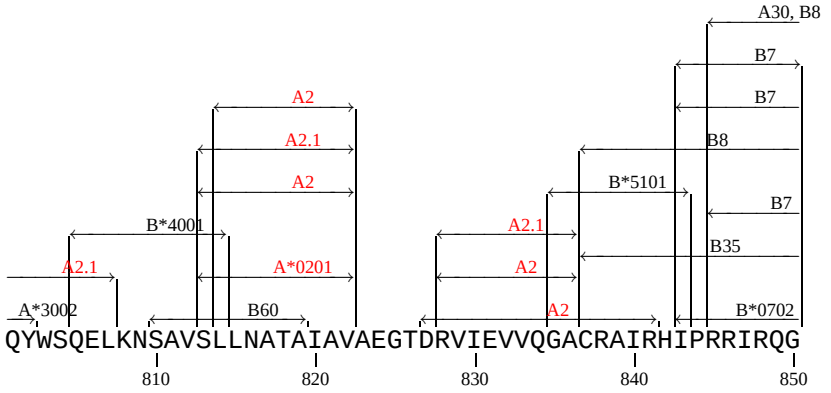

B8
B*0801
A*2402
Cw7
B27
A*2402
A*2402
A*2402
B14, B*1402
B14
B*1402
B14
B51
B15
B*5101
DPw4.2
CD4+ CTL(DR-1)
QQNNLLRAIEAQQHLLQLTVWGIKQLQARILAVERYLKDQQLLGIWGCSG
560
570
580
590
600


A*3002
A32
A2
A2
VLSIVNRVRQGYSPLSFQTHLPTPRGPDRPEGIEEEGGERDRDRSIRLVN
710
720
730
740
750


A*6802
A31
A3
A*3101
A*0301
A*2402
A31
A*3002
B27
B27
B*2705
A2.1
A2
GSLALIWDDLRSLCLFSYHRLRDLLLIVTRIVELLGRRGWEALKYWWNLL
760
770
780
790
800


B35
B*3501
KLICTTAVPWNASWSNKSLEQIWNHTTWMEWDREINNYTSLIHSLIEESQ
610
620
630
640
650

A2.1
A*2402
A2.1
A2
NQQEKNEQELLELDKWASLWNWFNITNWLWYIKLFIMIVGGLVGLRIVFA
660
670
680
690
700


A30, B8
B7
A2
B7
A2.1
B8
A2
B*5101
B*4001
A2.1
B7
A2.1
B35
A*3002
A*0201
A2
B60
A2
B*0702
QYWSQELKNSAVSLLNATAIAVAEGTDRVIEVVQGACRAIRHIPRRIRQG
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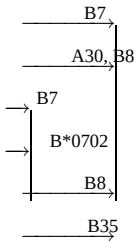
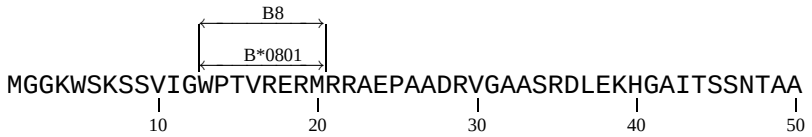
830
840
850

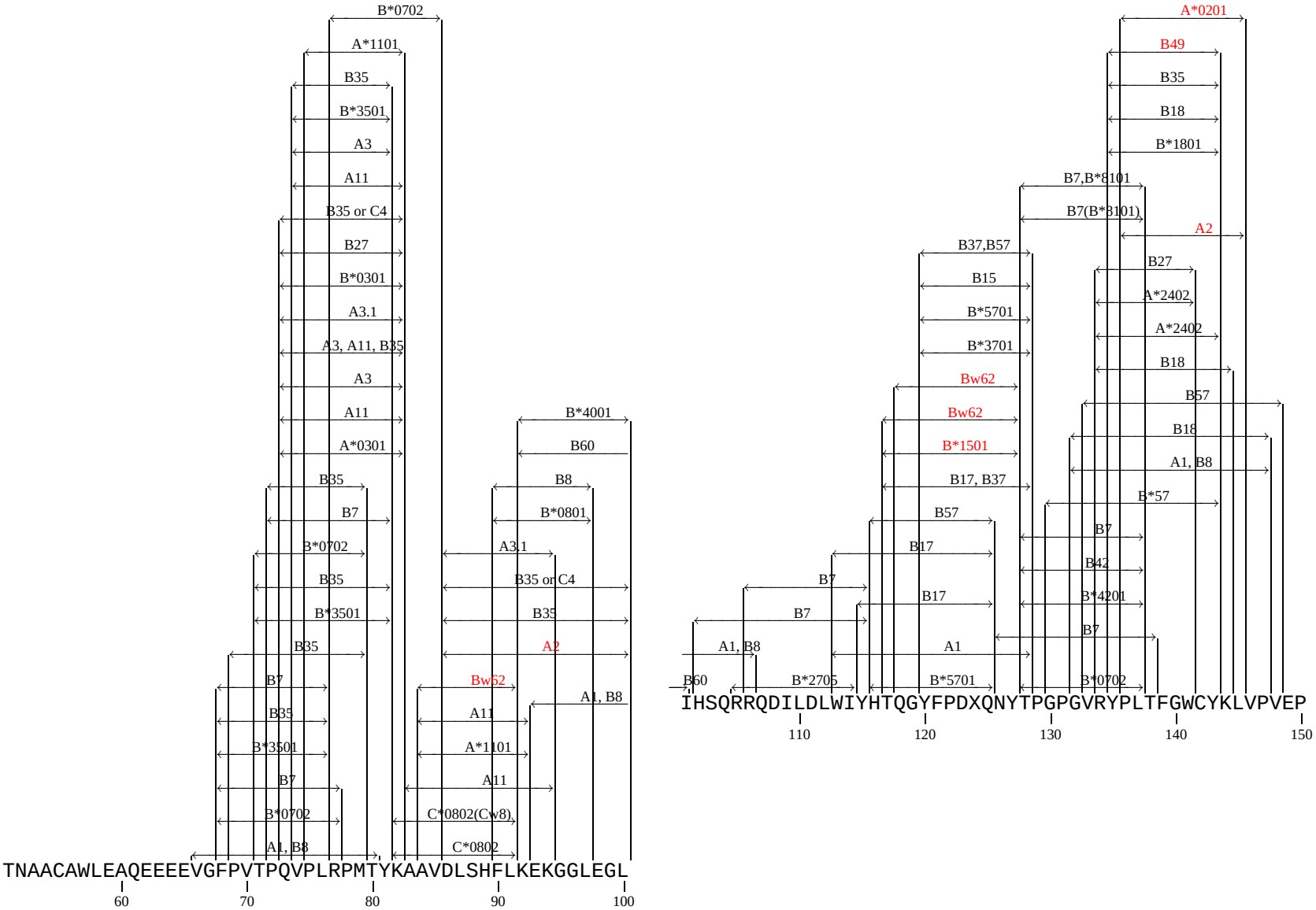


LERILL

-> gp41 end

Nef CTL Map

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B35
B*3501
A3
A11
B35 or C4
B27
B*0301
A3.1
A3, A11, B35
A3
A11
A*0301
B35
B7
B*0702
B35
B*3501
B35
B7
B*0702
A1, B8
B*4001
B60
B8
B*0801
A3.1
B35 or C4
B35
A2
Bw62
A1, B8
A11
A*1101
A11
C*0802(Cw8)
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B17
B42
B7
B17
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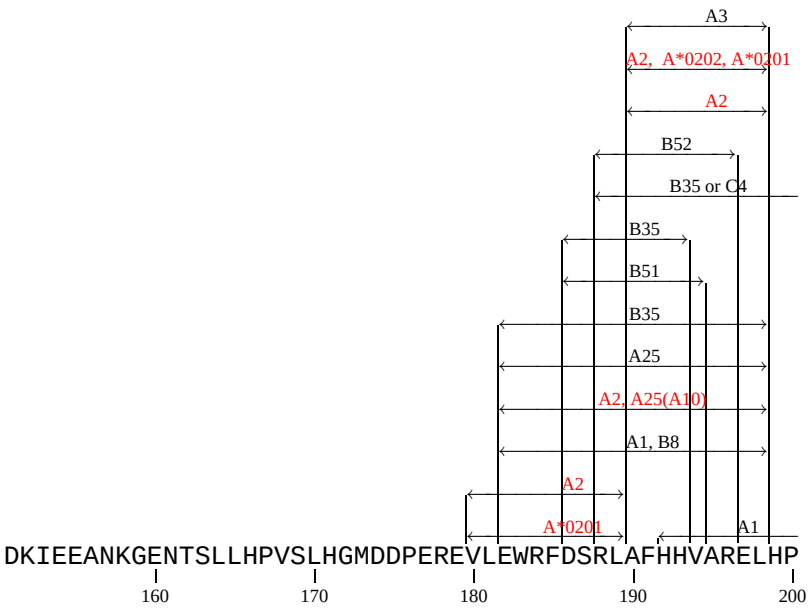

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[Rowland-Jones (1999)] S. L. Rowland-Jones, T. Dong, L. Dorrell, G. Ogg, P. Hansasuta, P. Krausa, J. Kimani, S. Sabally, K. Ariyoshi, J. Oyugi, K. S. MacDonald, J. Bwayo, H. Whittle, F. A. Plummer, & A. J. McMichael. Broadly cross-reactive HIV-speci£c cytotoxic T lymphocytes in highly-exposed persistently seronegative donors. *Immunol Lett* **66**:9–14, 1999. (Medline: 99217678).

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